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This document is an evaluation and assessment of scientific information relative to determining the health and welfare effects associated with exposure to various concentrations of nitrogen oxides in ambient air. The document is not intended as a complete, detailed literature review. It does not cite every published article relating to carbon monoxide and its effects in the environment. The literature through 1978 has been reviewed thoroughly for information relative to criteria. The major gaps in our current knowledge, relative to criteria, have been identified.

Though the emphasis is on the presentation of data on health and welfare effects, other scientific data are presented and evaluated in order to provide a better understanding of the pollutants in the environment. To this end, separate chapters concerning the properties and principles of formation, emissions, analytical methods of measurement, observed ambient concentrations, the global cycle, effects on vegetation and microorganisms, mammalian metabolism, effects on experimental animals, and effects on humans are included.

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NOTICE

This document is a draft. It has not been formerly released by EPA and should not at this stage be be cited or quoted.

FOREWORD

This document has been prepared in compliance with Section 108(c) of the Clean Air Act, 42 U.S.C. §7408 (c), which requires that the Administrator from time to time review, and, where appropriate, revise and reissue criteria. This document has also been prepared in compliance with Section 109(d)(1) of the Act, 42 U.S.C. §7409(d)(1) which requires that by December 31, 1980, the Administrator thoroughly review and, where appropriate, revise criteria in accordance with Section 108. Section 108(a)(2) requires that upon issuance the criteria reflect accurately the latest scientific information available relative to all identifiable effects on the public health or welfare from the presence of the pollutant in the ambient air.

These health and welfare criteria fulfill the regulatory purpose of serving as the basis upon which the Administrator will consider promulgation of a short-term (three hours or less) standard for nitrogen dioxide, and standards for other nitrogenous compounds (nitric and nitrous acids, nitrites, nitrates, nitrosamines and certain other derivatives of oxides of nitrogen). Criteria related to nitrogen dioxide will also be used to determine the need to promulgate a revised annual standard for nitrogen dioxide. The proposed standards are being published concurrently with the publication of this criteria document.

Although the preparation of a criteria document requires a comprehensive review and evaluation of the current scientific knowledge regarding the air pollutant in question. The references cited do not constitute a complete bibliography. The objective is to evaluate the scientific data base and to formulate criteria which may serve as the basis for decisions regarding the promulgation of national ambient air quality standards for the subject compounds. Scientific work of marginal significance to this end may not be included in the document.

The Agency is pleased to acknowledge the efforts and contributions of all persons and groups who have participated as authors or reviewers to this document. In the last analysis, however, the Environmental Protection Agency is responsible for its content.

PREFACE

This criteria document focuses on a review and assessment of the effects on human health and welfare of the nitrogen oxides, nitric oxide (NO) and nitrogen dioxide (NO_2), and the related compounds, nitrites, nitrates, nitrogenous acids, and nitrosamines. Although the emphasis is on presentation of health and welfare effects data, other scientific data are presented in order to provide a better understanding of these pollutants in the environment. To this end, separate chapters are included which discuss the nitrogen cycle, sources and emissions, atmospheric chemical processes which transform emissions of nitrogen oxides into related airborne compounds, transport and removal processes, measurement methods, and atmospheric concentrations of nitrogenous pollutants.

Of the oxides of nitrogen which occur in the atmosphere, NO_2 is the compound of most concern for human health. Controlled human exposure studies indicate that in some subjects increases in airway resistance of the pulmonary system are produced by short exposure to NO_2 concentrations in the range of 1300 to 3760 $\mu g/m^3$ (0.7 to 2.0 ppm). Studies of population exposure have demonstrated increased incidences of acute respiratory illness in young children associated with combustion products from gas stoves of which NO_2 is a significant component, but the exact effective concentrations and exposure times are difficult to determine.

Animal studies provide support for the mechanism of increased susceptibility to some respiratory pathogens as a consequence of NO_2 exposure but cannot provide quantitative data. Increases in animal susceptibility due to repeated short-term exposure eventually are as large as those produced by continuous exposure to the same concentration.

Many other effects have been shown to occur in animals at exposure concentrations comparable to those causing increases in susceptibility to respiratory infections. Usually, these have not been demonstrated in humans, but the potential for their occurrence cannot be ignored.

Air pollution degrades the appearance of distant objects and reduces the range at which they can be distinguished from the background. Nitrogen dioxide does not significantly reduce visual range but can be responsible for a portion of the brownish coloration observed in polluted air. NO_2 acts as a blue-minus filter for transmitted light. The strength of this filter effect is theoretically determined by the integral of NO_2 concentration along the sight path. An NO_2 concentration-times-distance product of less than 0.1 ppm-km NO_2 is sufficient to produce a color shift which is distinguishable in carefully-controlled laboratory tests. However, empirical observations under a variety of conditions are needed to determine the perceptibility of NO_2 in ambient air.

Nitrogen dioxide and particulate nitrates may also contribute to pollutant haze. The discoloration of the horizon sky due to NO_2 absorption is determined by the relative concentrations of NO_2 and light-scattering particles. At a visual range of 100 km, typical of the rural great plains area of the United States, as little as 0.003 ppm (6 $\mu g/m^3$) NO_2 can color the horizon noticeably. At a visual range of 10 km, typical of urban haze, at least 0.03 ppm (60 $\mu g/m^3$) NO_2 would be required to produce the same effect. Estimates of the possible role played by particulate nitrates are currently hampered by the lack of high quality data on their concentrations in ambient air.

Oxides of nitrogen as a class are major precursors to acidic precipitation which is defined as rainwater or snow having a pH of less than 5.6, the minimum expected from atmospheric carbon dioxide. Currently, the annual average pH of precipitation in the northeastern United States is between 4.0 and 5.0, and average pH values around 4.5 have been reported as far south as northern Florida. The pH of individual rain events may be as low as 2.2 to 3.0. Data, based on computations from chemical analyses of rain, indicate that the area affected by acidic rainfall has grown significantly over the past 20 years. In the United States, sulfuric and nitric acids make the primary contributions to the acidity in precipitation. There is strong evidence that the role of the nitrate ion has become increasingly important in recent years.

A number of direct effects of acidic precipitation on both terrestrial and aquatic biota have been reported. The effects include tissue damage and physiological impairment in plants, lethal effects on fish, and possible impacts on host-parasite or pathogenic processes. These effects may occur at specific short periods during an organism's life cycle, or may develop after repeated exposure. The ecological consequences of effects on specific terrestrial organisms or on the quality of soils have not been well measured. The long-term effects of acidification on aquatic ecosystems are widespread, regionally and globally, and can include decimation of fish populations. Little is known about the recovery of ecosystems from such effects, but liming of soils and lakes has been successful in a limited number of cases.

Oxides of nitrogen have also been shown to affect vegetation adversely. When crops are exposed to nitrogen dioxide alone in controlled studies, the ambient concentrations producing measurable injury are above those normally occurring in this country. Exceptions to this generality have been observed: the growth of Kentucky bluegrass was reduced about 25 percent by exposures to 210 $\mu g/m^3$ (0.11 ppm) NO_2 for 103.5 hours per week for 20 weeks during the winter months. A number of controlled studies on mixtures of nitrogen dioxide with sulfur dioxide show effects greater, in some cases much greater, than those effects caused by the individual pollutants alone. Some leaf injury to pinto bean, radish, soybean, tomato, oat and tobacco occurred after 4-hour exposure to 280 $\mu g/m^3$ (0.15 ppm) NO_2 in combination with 260 $\mu g/m^3$ (0.1 ppm) SO_2. Similar results were observed in green peas and swiss chard.

Kentucky bluegrass showed reductions in yield parameters ranging from 30 to 90 percent upon exposure for 20 weeks to 210 $\mu g/m^3$ (0.11 ppm) NO_2 in combination with 290 $\mu g/m^3$ (0.11 ppm) SO_2 for 103.5 hours per week.

Nitrogen dioxide has been found to cause deleterious effects on a wide variety of textile dyes and fabrics, plastics, and rubber. Significant fading of certain dyes on cotton and rayon has been shown after 12 weeks of exposure to 94 $\mu g/m^3$ (0.05 ppm) NO_2 at high humidity and temperature (90 percent, 90°F). Similar effects were obtained under similar conditions for various dyes on nylon, at 188 $\mu g/m^2$ (0.1 ppm). Yellowing of several white fabrics has been shown in exposure to 376 $\mu g/m^3$ (0.2 ppm) for 8 hours. Nitrates and nitrogenous acids have been implicated as possible causative and/or accelerating agents in the wet corrosion of metals and deterioration of electrical contracts.

ABSTRACT

This document is an evaluation and assessment of scientific information relative to determining the health and welfare effects associated with exposure to various concentrations of nitrogen oxides in ambient air. The document is not intended as a complete, detailed literature review. It does not cite every published article relating to carbon monoxide and its effects in the environment. The literature through 1978 has been reviewed thoroughly for information relative to criteria. The major gaps in our current knowledge, relative to criteria, have been identified.

Though the emphasis is on the presentation of data on health and welfare effects, other scientific data are presented and evaluated in order to provide a better understanding of the pollutants in the environment. To this end, separate chapters concerning the properties and principles of formation, emissions, analytical methods of measurement, observed ambient concentrations, the global cycle, effects on vegetation and microorganisms, mammalian metabolism, effects on experimental animals, and effects on humans are included.

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ABBREVIATIONS, ACRONYMS, AND SYMBOLS

Å	Ångstrom (10^{-10} meter)
"A" strain	A particular type of influenza virus
$AaDO_2$	Difference between alveolar and arterialized partial pressure of oxygen
AAS	Atomic absorption spectroscopy
AATCC	American Association of Textile Chemists and Colorists
Ad	A particular strain of laboratory mouse
AICHE	American Institute of Chemical Engineers
AM	Alveolar macrophage
AMP	Adenosine monophosphate; adenosine 5' phosphate
ANSA	8-anilino-1-naphthalene-sulfonic acid
APCD	Air Pollution Control District
APHA	American Public Health Association
A/PR/8	A particular strain of influenza virus
A/PR/8/34	A particular strain of influenza virus
AQCR	Air Quality Control Region
AQSM	Air Quality Simulation Model
ASTM	American Society for Testing and Materials
atm	One atmosphere, a unit of pressure
ATP	Adenosine triphosphate
avg	Average
BAKI	Potassium iodide solution acidified with boric acid
BHA	Butylated hydroxyanisole
BHT	Butylated hydroxytoluene
BP	Blood pressure

b_{scat}	Extinction coefficient due to scatter by aerosols
C3H	A particular strain of laboratory mouse
C57BL	A particular strain of laboratory mouse
C57BL/6	A particular strain of laboratory mouse
cAMP	Cyclic adenosine monophosphate; adenosine 5'-phosphate
CAMP	Community Air Monitoring Program
CD-1	A particular strain of laboratory mouse
cGMP	Cyclic guanosine monophosphate; guanosine 5'-phosphate
°C	Degrees Celsius (Centigrade)
^{14}C	A radioactive form of carbon
CHE	Cholinesterase
C_L	Lung compliance
C_{Ldyn}	Dynamic lung compliance
C_{Lstat}	Static lung compliance
cm	Centimeter
CNS	Central nervous system; the brain and spinal cord
CO	Carbon monoxide
CO_2	Carbon dioxide
CoA	Coenzyme A
COH	Coefficient of haze
CPK	Creatine phosphokinase
CR-1	A particular strain of laboratory mouse
CRD	Chronic respiratory disease
CV	Closing volume
C x T	Exposure concentration in ppm multiplied by time of exposure in hours or other time measurement
d	Day

DEN	Diethylnitrosamine (also DENA)
DIFKIN	Diffusion Kinetics Model
DL_{CO}	Diffusion capacity of the lung for carbon monoxide
DMN	Dimethylnitrosamine
DNA	Deoxyribonucleic acid
D = CT	Dose equals concentration multiplied by time
DPPD	N,N diphenylphenylenediamine
EC	Prefix of International Commission on Enzymes' identification numbers
EKG	Electrocardiogram
EPA	U.S. Environmental Protection Agency
°F	Degrees Fahrenheit
FEF	Forced expiratory flow
FET	First-edge time
FEV	Forced expiratory volume
$FEV_{1.0}$	One-second forced expiratory volume
$FEV_{0.75}$	0.75-second forced expiratory volume
FRM	Federal Reference Method for air quality measurement
ft	Foot
FT	Fourier transform spectroscopy (also FS)
FVC	Forced vital capacity
g	Gram
G6P	Glucose-6-phosphate
GC	Guanylate cyclase
GL	Gas chromatography
GC-MS	Gas chromatograph in combination with mass spectrometry
GM	General Motors Corporation

GMP	Guanosine 5'-phosphate; guanosine monophosphate
GSH	A tripeptide, glutathione (reduced form)
GSSG	The disulfide (oxidized) form of GSH
$H^\cdot$	Hydrogen (free radical)
^3H	Tritium; a radioactive form of hydrogen
ha	Hectare
HbO_2	Oxyhemoglobin
HNO_2	Nitrous acid (also HONO)
HNO_3	Nitric acid (also $HONO_2$)
$HO^\cdot$	Hydroxyl free radical (also OH)
$HO_2^\cdot$	Hydroperoxyl free radical
HO_2NO	Pernitrous acid
HO_2NO_2	Pernitric acid (also $HOONO_2$)
hr	Hour
HR	Heart rate
hv	Planck's constant (h) times the frequency of radiated energy (v) = Quanta of energy (E)
H_2O_2	Hydrogen peroxide
H_2S	Hydrogen sulfide
H_2SO_4	Sulfuric acid
IARC	International Agency for Research on Cancer
Ig	Immunoglobulins
IgA	Immunoglobulin A fraction
IgG	Immunoglobulin G fraction
IgG_1	Immunoglobulin G_1 fraction
IgG_2	Immunoglobulin G_2 fraction
IgM	Immunoglobulin M fraction

in	Inch
IR	Infrared
k	Rate constant or dissociation constants
kg	Kilograms
km	Kilometer
l	Liter (also ℓ)
LC50	Lethal concentration 50%; that concentration which is lethal to 50 percent of test subjects
LD50	Lethal dose 50%; dose which is lethal to 50 percent of the subjects
LT50	The time required for 50 percent of the test animals to die when given a lethal dose
LDH	Lactic acid (lactate) dehydrogenase
LPS	Bacterial lipopolysaccharide
m	Meter
M	Molar
M	Third body (in a reaction)
MAK	Maximum permissible concentration (in Germany)
max	Maximum
MFR	Maximal flow rate
$\mu g/m^3$	Micrograms per cubic meter
mg/m^3	Milligrams per cubic meter
Mg	Magnesium
ml	Milliliter
mM	Millimoles
MMD	Mass median diameter
MMFR	Mid-maximal flow rate
mo	Month

MPC	Maximum permissible concentration (in the U.S.S.R.)
MT	Metric Ton
N	Nitrogen
N	Normal
^{13}N	A radioactive form of nitrogen
N-6-MI	N-nitrosoheptamethyleneimine
NA	Not applicable
NAAQS	National Ambient Air Quality Standard
$NaCl$	Sodium chloride; common table salt
NAD^+	Nicotinamide-adenine dinucleotide (+ indicates oxidized form)
NADB	National Air Data Bank
NADH	Nicotinamide-adenine dinucleotide (reduced form)
NADPH	Nicotinamide-adenine dinucleotide phosphate (reduced form)
$NaOH$	Sodium hydroxide
NAS	National Academy of Sciences
NASN	National Air Surveillance Network
NDIR	Nondispersive infrared
NEDA	N-(1-Naphthyl)-ethylenediamine dihydrochloride
NEDS	National Emissions Data System
NEIC	National Enforcement Investigations Center
ng	Nanogram
NH_4	Ammonium ion or radial
nm	Nanometer
NO	Nitric oxide
NOHb	Nitrosylhemoglobin

NO_x	Nitrogen oxides
N_2O	Nitrous oxide
NO_2	Nitrogen dioxide
N_2O_3	Dinitrogen trioxide
N_2O_4	Dinitrogen tetroxide
NSF	National Science Foundation
O	Atomic oxygen
$O(^1D)$	Excited atomic oxygen
O_3	Ozone
OH	Hydroxyl group
$O(^3P)$	Ground state atomic oxygen
^{32}P	A radioactive form of phosphorus
$PaCO_2$	Alveolar partial pressure of carbon dioxide
$PaCO_2$	Arterial partial pressure of carbon dioxide
PAH	p-Ammiohippuric acid
PAN	Peroxyacetyl nitrate
PaO_2	Arterial partial pressure of oxygen
PAO_2	Alveolar partial pressure of oxygen
pH	Log of the reciprocal of the hydrogen ion concentration
PHA	Phytohemagglutinin
PO_2	Partial oxygen pressure
ppb	Parts per billion
pphm	Parts per hundred million
ppm	Parts per million
ppt	Parts per trillion
Q	Cardiac output
QRS	A complex of three distinct electrocardiogram waves which represent the beginning of ventricular contraction

RAMS	Regional Air Monitoring System
RAPS	Regional Air Pollution Study
R_{aw}	Airway resistance
RBC	Red blood cell; erythrocyte
RM	Reference method for air quality measurement
RNA	Ribonucleic acid
RV	Residual volume
SAI	Science Applications, Inc.
SD	Standard deviation
SGOT	Serum glutamic-oxaloacetic transaminase
SGPT	Serum glutamic-pyruvic transaminase
SH-	Sulfhydryl group
SMSA	Standard Metropolitan Statistical Area
SN	Suspended nitrates
SO_2	Sulfur dioxide
SPF	Specific pathogen free
SR_{aw}	Specific airway resistance
SRM	Standard reference material
SS	Suspended sulfates
STP	Standard temperature and pressure
TEA	Triethanolamine
Tg	Terragram; 10^6 metric tons or 10^{12} grams
TGS-ANSA	A 24-hour method for the detection of analysis of NO_2 in ambient air
TLC	Total lung capacity
TPTT	20 percent transport time
TSP	Total suspended particulate

USEPA	U.S. Environmental Protection Agency
UV	Ultraviolet radiation
VC	Vital capacity
VE	Ventilatory volume
VEE	Venezuelan equine encephalomyelitis (virus)
$\dot{V}_{max}$	Maximum expiratory flow rate
V_T	Total volume
V/V	Volume per volume
WBC	White blood cells
wk	Week
yr	Year
Zn	Zinc
Mg	Microgram
Ml	Microliter
Mm	Micrometer
>	Greater than
<	Less than
~	Approximately

CONTRIBUTORS AND REVIEWERS

Principal authors:

Dr. Charles E. Anderson, Department of Botany, North Carolina State University, Raleigh, North Carolina.

Dr. Paul J. Crutzen, National Center for Atmospheric Research, Boulder, Colorado.

Dr. Sandor J. Freedman, System Sciences, Inc., Chapel Hill, North Carolina.

Dr. J. H. B. Garner, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Judith A. Graham, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Daniel Grosjean, Statewide Air Pollution Research Center, University of California, Riverside, California.

Dr. Steven M. Horvath, Institute of Environmental Stress, University of California at Santa Barbara, Santa Barbara, California.

Dr. Gory J. Love, Consultant, System Sciences, Inc., Chapel Hill, North Carolina.

Dr. Daniel B. Menzel, Department of Pharmacology, Duke University, Durham, North Carolina.

Dr. Joseph Roycroft, Jr., Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Victor S. Salvin, University of North Carolina, Greensboro, North Carolina.

Dr. John H. Seinfeld, Department of Chemical Engineering, California Institute of Technology, Pasadena, California.

Dr. John C. Trijonis, Technology Service Corporation, Santa Fe, New Mexico.

Dr. Warren H. White, Technology Service Corporation, Pasadena, California.

Contributing authors and reviewers:

Dr. Martin Alexander, Department of Agronomy, Cornell University, Ithaca, New York.

Dr. Patrick L. Brezonik, Department of Environmental Engineering Sciences, University of Florida, Gainesville, Florida.

Dr. John B. Clements, Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. T. Timothy Crocker, Department of Community and Environmental Medicine, University of California, Irvine, California.

Dr. Basil Dimitriades, Environmental Sciences Research Laboeatory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. George M. Duggan, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Richard Ehrlich, Illinois Institute of Technology Research Institute, Chicago, Illinois.

Mr. Robert B. Faoro, Monitoring and Data Analaysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. C. Eugene Feigley, Department of Environmental Health, University of South Carolina, Columbia, South Carolina.

Dr. Jack Fishman, National Center for Atmospheric Research, Boulder, Colorado.

Dr. Robert Frank, Department of Environmental Health, University of Washington, Seattle, Washington.

Dr. Elliot Goldstein, Department of Internal Medicine, University of California, Davis, California.

Dr. Lester D. Grant, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Mark M. Greenberg, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Jack D. Hackney, Rancho Los Amigos Hospital Campus, University of Southern California, Downey, California.

Mr. Albert V. Hardy, System Sciences, Inc., Chapel Hill, North Carolina.

Dr. Steven M. Horvath, Institute of Environmental Stress, University of California, Santa Barbara, California.

Dr. Harvey E. Jeffries, Department of Environmental Sciences and Engineering, University of North Carolina, Chapel Hill, North Carolina.

Dr. Evaldo Kothny, California State Department of Health, Berkeley, California.

Mr. William S. Lanier, Industrial Environmental Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. David J. McKee, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Sidney S. Mirvish, Eppley Institute for Research in Cancer, University of Nebraska, Omaha, Nebraska.

Mr. Larry J. Purdue, Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Carl M. Shy, Institute of Environmental Health, University of North Carolina, Chapel Hill, North Carolina.

Mr. Herschel H. Slater, PEDCo Environmental, Inc., Durham, North Carolina.

Mr. Mark G. Smith, System Sciences, Inc., Chapel Hill, North Carolina.

Dr. Edward P. Stahel, Department of Chemical Engineering, North Carolina State University, Raleigh, North Carolina.

Dr. David T. Tingey, Corvallis Environmental Research Laboratory, U.S. Environmental Protection Agency, Corvallis, Oregon.

Reviewers:

Mr. Gerald G. Akland, Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Aubrey P. Altschuller, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Mark R. Antell, Division of Stationary Source Enforcement, U.S. Environmental Protection Agency, Washington, D.C.

Mr. John D. Bachmann, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Ronald L. Baron, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Norman J. Beloin, Surveillance and Analysis Division, Region I, U.S. Environmental Protection Agency, Boston, Massachusetts.

Mr. Michael A. Berry, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Frank M. Black, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Ms. Frances P. Bradow, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Joseph J. Bufalini, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Ronald C. Campbell, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Angelo P. Capparella, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Capt. Harvey J. Clewell, III, Civil and Environmental Engineering Development Office, Tyndall Air Force Base, Florida.

Mr. Stanton Coerr, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. David L. Coffin, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Henry S. Cole, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Ellis B. Cowling, Department of Plant Pathology, North Carolina State University, Raleigh, North Carolina.

Mr. Swep T. Davis, Deputy Assistant Administrator for Water Planning and Standards, Office of Water and Waste Management, U.S. Environmental Protection Agency, Washington, D.C.

Dr. Kenneth L. Demerjian, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Marcia C. Dodge, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Thomas G. Dzubay, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Thomas G. Ellestad, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Douglas Fennell, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Neil H. Frank, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Gustave Freeman, Stanford Research Institute, Menlo Park, California.

Dr. Donald E. Gardner, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, N.C.

Dr. Philip L. Hanst, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Fred H. Haynie, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Walter W. Heck, Department of Botany, North Carolina State University, Raleigh, North Carolina.

Mr. George C. Holzworth, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Allen Hoyt, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. F. Gordon Hueter, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Michael H. Jones, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. John H. Knelson, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Kenneth T. Krost, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Ms. Pamela A. Mealer, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Thomas R. McCurdy, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Thomas B. McMullen, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. M. Rodney Midgett, Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Edwin L. Meyer, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. William Nelson, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Stephen Nesnow, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. John R. O'Connor, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Joseph Padgett, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Dennis J. Reutter, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Harvey M. Richmond, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Harold G. Richter, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Ms. Karen Rourke, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Joseph Seitler, Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C.

Mr. Donald H. Sennett, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Larry L. Sims, Air Surveillance and Investigation Section, Region X, U.S. Environmental Protection Agency, Seattle, Washington.

Mr. James R. Smith, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Carl R. Sova, Air Engineering Branch, Region IV, U.S. Environmental Protection Agency, Atlanta, Georgia.

Mr. Robert K. Stevens, Environmental Sciences Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Orin W. Stopinski, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Joseph Suggs, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Mr. Matthew Van Hook, Air, Noise and Radiation Division, U.S. Environmental Protection Agency, Washington, D.C.

Dr. David E. Weber, Corvallis Environmental Research Laboratory, U.S. Environmental Protection Agency, Corvallis, Oregon.

Mr. Albert H. Wehe, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Dr. Jerome Weslowski, California State Department of Health, Berkeley, California.

Mr. Larry Zaragoza, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Enviornmental Protection Agency, Research Triangle Park, North Carolina.

Project management, editing, production, and word processing at System Sciences, Inc., Chapel Hill, North Carolina, under contract to the Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina:

Dr. Edgar A. Parsons
Ms. Gayla Benignus
Dr. Sandor J. Freedman
Ms. Doris M. Lange
Ms. Elsa M. Lewis-Heise
Ms. Jeannine A. McFarland
Ms. Trudy E. Oakes
Ms. Diane Robinson
Ms. E. L. Rusten
Mr. Mark G. Smith
Mr. Robert E. Wenzel

Word processing and other assistance at the Environmental Criteria and Assistance Office, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina:

Ms. Dela Bates
Ms. Diane Chappell
Ms. Constance Van Oosten
Ms. Evelynne Rash
Ms. Donna Wicker

CLEAN AIR SCIENCE ADVISORY COMMITTEE
SUBCOMMITTEE ON AIR QUALITY
CRITERIA FOR OXIDES OF NITROGEN

Dr. Mary O. Amdur
Department of Nutrition and Food Science
Massachusetts Institute of Technology
Cambridge, MA

Dr. Domingo M. Aviado
Allied Chemical
Morristown, NJ

Dr. Judy A. Bean
Associate Professor
Department of Preventive Medicine
College of Medicine
University of Iowa
Iowa City, IA

Dr. Robert Dorfman
Department of Economics
Harvard University
Cambridge, MA

Dr. Sheldon K. Friedlander
Professor of Chemical Engineering and
Environmental Health Engineering
W. M. Keck Laboratory
California Institute of Technology
Pasedena, CA

Mr. Harry H. Hovey, Jr.
New York State, Department of
Environmental Conservation
Albany, NY

Dr. Donald H. Pack
Consulting Meteorologist
McLean, VA

1. SUMMARY OF EFFECTS OF OXIDES OF NITROGEN AND RELATED COMPOUNDS ON HUMAN HEALTH AND WELFARE

1.1 INTRODUCTION

This criteria document critically evaluates scientific information on both short- and long-term health and welfare effects of nitrogen oxides as well as other nitrogenous compounds, such as nitric and nitrous acids, nitrites, nitrates, and nitrosamines. Pursuant to Sections 108 and 109 of the Clean Air Act, as amended in 1977, it is to serve as a basis for evaluating the need to promulgate National Ambient Air Quality Standards for any or all of these compounds. The scientific evidence reviewed includes significant new research published since the first edition of Air Quality Criteria for Nitrogen Oxides in 1971.

Major questions addressed in this document include the following:

(1) What are identifiable health effects associated with exposure to airborne nitrogenous compounds?

(2) At what level(s) of exposure do such health effects occur in humans?

(3) Are there population subgroups especially susceptible to the effects of exposure to airborne nitrogenous compounds?

(4) What are the major welfare effects on visibility, vegetation, and materials associated with airborne nitrogenous compounds?

(5) At what concentration levels do effects on visibility, vegetation and materials occur?

(6) To what degree do airborne nitrogenous compounds contribute to large-scale environmental effects such as acidic precipitation?

(7) Are presently available techniques for measuring atmospheric levels of nitrogenous compounds adequate?

(8) What are the major sources of airborne nitrogenous compounds?

(9) What concentrations of nitrogenous compounds of concern occur in ambient air?

This present chapter summarizes available data on the effects on human health and welfare of nitrogen oxides (NO_x), consisting principally of nitrogen dioxide (NO_2) and nitric oxide (NO), and other nitrogenous compounds which may be derived from NO_x through atmospheric transformations. Since NO_2 has been most conclusively demonstrated to exert deleterious effects, the emphasis here has been placed on the interpretation of data bearing on NO_2 effects in order to estimate ambient air concentration levels at which such effects on human health and welfare occur. Information on natural and man-made sources, atmospheric chemical and physical transformations, techniques of sampling and analysis, and additional data on ambient concentrations are also summarized in the present chapter. The reader is referred to the subsequent document chapters for more detailed information on all of the above topics.

1.2 SOURCES, TRANSFORMATIONS AND AMBIENT LEVELS OF NITROGEN OXIDES

Oxides of nitrogen have their origin in a number of natural and man-made processes (Chapters 4 and 5). In terms of sources giving rise to significant human exposure, however, the most important emissions occur as a result of man's burning of fossil fuels such as coal, oil or gasoline. Emissions from motor vehicles (mobile combustion) and from installations burning fossil fuels (stationary combustion) are the two largest sources, constituting about 44 percent and 51 percent of the nationwide NO_x emissions, respectively, in 1976. In the stationary combustion category, electric utilities were responsible for approximately 56 percent of the NO_x emissions and industrial combustion accounted for another 38 percent. In the mobile combustion category, highway vehicle emissions constituted about 77 percent, with the rest attributable to non-highway vehicles.

In most ambient situations, NO_2 is not emitted directly into the atmosphere in significant amounts (typically less than 10 percent of NO_x emissions). It is important to note, however, that NO_2 forms upwards of 30 to 50 percent of the total NO_x emissions from certain diesel and jet turbine engines under specific load conditions. Diesel emissions of NO_2 may become of concern in local situations if there is a widespread increase in the use of diesel-powered vehicles. NO_2 is formed, generally, from the oxidation of the more commonly emitted compound nitric oxide (NO). The chemical pathways by which NO is transformed to NO_2 are complex and involve other atmospheric constituents such as hydrocarbons and ozone. Also, NO_2 is not the final product of atmospheric reactions. It may decompose in the presence of sunlight or it may undergo further transformation into gaseous nitric acid (HNO_3) and/or nitrate aerosols, small particles suspended in ambient air (Chapter 6). For these and other reasons, the relationship between NO_x emissions and resulting ambient NO_2 concentrations is neither direct nor constant.

Oxides of nitrogen and their atmospheric transformation products may be transported in ambient air over distances ranging up to hundreds of kilometers from the emissions source and over times ranging up to several days. Ultimate removal from the air occurs by a variety of processes including uptake by vegetation, deposition on surfaces and precipitation by rain or snow. The times and distances involved in the transformation, transport, and removal of atmospheric nitrogenous compounds indicate that these pollutants not only exert an impact in proximity to primary sources, but are also of concern in relation to deleterious effects exerted at considerable distances from points of initial emission or transformation. The contribution of nitric acid to the phenomenon of acidic precipitation, which may occur hundreds of kilometers from a source or sources of NO_x, is an example of such a non-local impact (Chapter 11).

It has been suggested that oxides of nitrogen may react in the atmosphere with amines emitted by certain sources to produce nitrosamines. However, there is little evidence to date to indicate that this reaction takes place in ambient situations or that the atmospheric route for human exposure to this class of compounds is a cause for concern.

In general, adequate methodology now exists for sampling and analysis of NO_2 concentrations in ambient air (Chapter 7). Techniques for routine determination of atmospheric concentrations of nitric acid and nitrate aerosols, however, are currently only in the developmental stage.

Various government agencies have routinely monitored NO_2 for some time. The following summary of recent ambient levels of NO_2 occurring nationwide is given to place in perspective the concentration levels associated with the health and welfare effects discussed later in this chapter. Chapter 8 contains a more thorough survey of available monitoring data. The reader should note that not all persons living or working in the areas mentioned will actually experience the NO_2 concentrations cited. Moreover, considering the likelihood that fixed monitoring sites may not intercept the maximum NO_2 concentration, there is a possibility that the concentrations reported underestimate the exposure experienced by at least some portion of the population in the areas cited.

Examination of selected nationwide monitoring data for 1975 to 1980 reveals that during at least one of these years, peak 1-hour NO_2 concentrations equalling or exceeding 750 $\mu g/m^3$ (0.4 ppm) were experienced in Los Angeles and several other California sites; Ashland, Kentucky; and Port Huron, Michigan. Additional sites reporting at least one peak hourly concentration equalling or exceeding 500 $\mu g/m^3$ (0.27 ppm) include: Phoenix, Arizona; St. Louis, Missouri; New York City, New York; 14 additional California sites; Springfield, Illinois; Cincinnati, Ohio; and Saginaw and Southfield, Michigan. Other scattered sites, distributed nationwide, reported maxima close to this value. Recurrent NO_2 hourly concentrations in excess of 250 $\mu g/ m^3$ (0.14 ppm) were quite common nationwide in 1975 to 1980.

Annual arithmetic means for NO_2 concentrations in 1976 exceeded 100 $\mu g/m^3$ (0.053 ppm) at Anaheim, El Cajon, Riverside, San Diego, and Temple City, California. Other sites reporting yearly arithmetic means for 1976 equalling or exceeding 100 $\mu g/m^3$ (0.053 ppm) included Chicago, Illinois, and Southfield, Michigan. However, virtually none of the same monitoring sites still operating in 1980 reported values above 100 $\mu g/m^3$ in that year (except for one in San Diego; 114 $\mu g/m^3$).

1.3 EFFECTS OF NITROGEN OXIDES ON HUMAN HEALTH

As summarized above, nitric oxide (NO) is the most prevalent oxide of nitrogen directly emitted into the ambient air as the result of anthropogenic activities such as fossil fuel combustion. In addition, other compounds, such as nitrogen dioxide (NO_2), gaseous nitric acid (HNO_3), nitrites and nitrate aerosols, have been clearly established as being formed in the ambient air as the result of atmospheric chemical reactions of NO and other nitrogen oxides with non-nitrogenous substances. The formation of nitrosamines in the ambient air has been suggested, but not convincingly demonstrated.

Concern has been expressed about possible harmful health effects of virtually all of the above types of nitrogen oxide compounds. Despite such concern and considerable scientific inquiry on the subject, there now exist relatively little hard data linking specific health effects to the majority of the above nitrogen oxide compounds. The one notable exception is nitrogen dioxide.

The literature reviewed in detail in Chapter 15 of this document indicates that nitric oxide (NO) is not of direct concern for human health effects at typical ambient air concentration levels recorded over U.S. cities. Similarly, there is presently no evidence that nitrites at levels ordinarily found in the atmosphere are of concern for human health. In addition, although it has been suggested that atmospheric nitrates may be associated with increased numbers of asthmatic attacks, no data are presently available that are sufficient upon which to base firm conclusions regarding the subject.

The lack of strong evidence, or even conclusive pertinent studies, associating health effects with most nitrogen oxide compounds is in striking contrast to the more extensive and convincing data base which links a number of specific health effects to nitrogen dioxide (NO_2). Information on NO_2 formation and its effects is, therefore, most heavily emphasized both in the present summary chapter and elsewhere in this document. In regard to the types of health effects of NO_2 most definitively characterized to date, the effects of NO_2 on the respiratory system have been most extensively delineated and appear to be of most concern in terms of both acute and long-term health implications. Major attention is accorded here to the summary and interpretation of key studies and the overall pattern of results bearing on respiratory system effects of NO_2.

1.3.1 Nitrogen Dioxide Respiratory System Effects

Nitrogen dioxide's effects on human and animal respiratory systems span a broad spectrum both in terms of initial severity and ultimate long-term health impact. The continuum of observed NO_2 effects ranges from (1) death or irreversible pulmonary damage seen with accidental high exposures to NO_2 primarily in occupational settings; through (2) less severe, but significant short-term and chronic tissue damage, functional impairment, and exacerbation of other disease processes observed at lower exposure levels; to (3) comparatively mild transient effects, such as impaired olfactory reception, which commence at still lower NO_2 levels.

Acute high level exposures to NO_2 that have occurred accidentally or in occupational settings demonstrate that concentrations in the range of 560,000 $\mu g/m^3$ (300 ppm) or higher are likely to result in rapid death. Concentrations in the range of 280,000 to 380,000 $\mu g/m^3$ (150-200 ppm) are not likely to cause immediate death, but severe respiratory distress and death occur after a period of 2 to 3 weeks. In such cases, the cause is almost always bronchiolitis fibrosa obliterans. Non-fatal acute exposures to 94,000 to 190,000 $\mu g/m^3$ (50 to 100 ppm) NO_2 are associated with reversible bronchiolitis, whereas acute exposures to 47,000 to 140,000 $\mu g/m^3$ (25 to 75 ppm) are associated with bronchitis or bronchial pneumonia and are usually but not always followed by essentially complete recovery. It should be noted that exposures to such high levels of NO_2 only typically occur in connection with certain occupational or accidental (e.g. fires) circumstances, and are not of concern in relation to ambient air exposures of the general public.

In addition to the above health effects induced by acute high level NO_2 exposures, however, a variety of respiratory system effects have been reported to be associated with exposures to lower concentrations of NO_2. Extensive literature characterizing such effects has resulted from three general approaches: (1) controlled human exposure studies; (2) human epidemiological studies; and (3) animal toxicology studies. The major types of NO_2-induced respiratory effects characterized by these different approaches include: (1) increased airway resistance (R_{aw}) and other indications of altered pulmonary function as observed in controlled clinical studies; (2) increased incidence of human respiratory illnesses, as determined by certain epidemiological studies; and (3) lung tissue damage and increased susceptibility to respiratory infection, as demonstrated in animal toxicology studies.

Collectively, these studies provide evidence indicating that certain human health effects may occur as the result of exposures to NO_2 concentrations approaching or falling within the range of recorded ambient air NO_2 levels. Key studies providing evidence for such effects are summarized and interpreted below in relation to several critical health issues. Those issues include: (1) consideration of lowest effective single NO_2 exposure level(s) inducing particular respiratory effects in healthy and sensitive human subjects; (2) assessment of lowest effective exposure levels at which repeated or intermittent NO_2 exposures produce effects in human populations; and (3) consideration of the relative significance of observed effects in terms of understanding the likely impact of ambient NO_2 exposures on human health.

1.3.1.1 Controlled Human Exposure Studies--Controlled human exposure (clinical) studies have generated extensive information on the lowest effective dose levels for the induction of respiratory effects by single short-term NO_2 exposures. Some studies have focussed on such effects in healthy adults; other studies have assessed respiratory effects in "sensitive" members of the population, e.g., individuals with chronic respiratory problems. Summarized in Table 1-1 are the most important controlled human exposure studies of short-term NO_2 exposure effects on pulmonary functions in healthy adult subjects.

The studies summarized in Table 1-1 indicate that increased airway resistance (R_{aw}) and other physiological changes suggesting impaired pulmonary function have been clearly demonstrated to occur in healthy adults with single 2-hr NO_2 exposures ranging from 3760 to 13,200 $\mu g/m^3$ (2.5 to 7.0 ppm). Certain studies also indicate that significant effects occur in healthy subjects with shorter (3-15 min) exposures to the same or possibly lower levels of NO_2 administered either alone or in combination with NaCl aerosol.

More specifically, in regard to the latter point, Suzuki and Ishikawa (1965) observed altered respiratory function after exposure of healthy subjects to NO_2 levels of 1300 to 3760 $\mu g/m^3$ (0.7 to 2.0 ppm) for 10 minutes. Their data however, preclude a clear association of observed effects with any particular concentration in the range of 1300 to 3760 $\mu g/m^3$ (0.7 to 2.0 ppm) NO_2 exposure.

Hackney et al. (1978) reported no statistically significant changes in any of the pulmonary functions tested with the exception of a marginal loss in forced vital capacity after exposure to 1880 $\mu g/m^3$ (1.0 ppm) NO_2 for 2 hours on two successive days (1.5% mean decrease, $P < 0.05$). The authors question the health significance of this small, but statistically significant change in forced vital capacity in healthy subjects and suggest that the changes found may be due to random variation.

TABLE 1-1. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION IN CONTROLLED STUDIES OF HEALTHY HUMAN ADULTS**

Concentration µg/m^3	ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference***
13,000	7.0	NO_2	Several	10-120 min.	Increased R_{aw}* in some subjects. Others tolerated 30,000 µg/m^3 (16 ppm) with no increase in R_{aw}.	Yokoyama, 1972
9,400	5.0	NO_2	11	2 hrs.	Increase in R_{aw}* and a decrease in $AaDO_2$* with intermittent light exercise. No enhancement of the effect when 200 µg/m^3 (0.1 ppm) O_3 and 13,000 µ/m^3 (5.0 ppm) SO_2 were combined with NO_2 but recovery time apparently extended.	von Nieding et al., 1977
9,400	5.0	NO_2	16	15 min.	Significant decrease in DL_{CO}*	von Nieding et al., 1973
9,400	5.0	NO_2	13	15 min.	Significant decrease in PaO_2* but end expiratory PO_2* unchanged with significant increase in systolic pressure in the pulmonary artery.	von Nieding et al., 1970
7,500 to 9,400	4.0 to 5.0	NO_2	5	10 min.	40% decrease in lung compliance 30 min. after exposure and increase in expiratory and inspiratory flow resistance that reached maximum 30 min. after exposure.	Abe, 1967
5,600	3.0	NO2	1	5 min.	Increase in R_{aw}* compared to pre-exposure values (enhanced by NaCl aerosol). More subjects were tested at higher exposures.	Nakamura, 1964
11,300	6.0	NO_2	1	5 min.		

TABLE 1-1. (continued)

Concentration		Pollu-tant	No. of Healthy Subjects	Exposure Time	Effects	Reference
µg/m³	ppm					
1,880	1.0	NO_2	8	2 hrs.	No increase in R_{aw}.	Beil and Ulmer, 1976
4,700	2.5	NO_2	8	2 hrs.	Increased R_{aw} with no further impairment at higher concentrations. No change in arterial PO_2 pressure or PCO_2 pressure.	
14,000	7.5	NO_2	16	2 hrs.	Increased sensitivity to a bronchoconstrictor (acetylcholine) at this concentration but not at lower concentrations.	
9,400	5.0	NO_2	8	14 hrs.	Increase in R_{aw} during first 30 min. that was reduced through second hour followed by greater increases measured at 6, 8 and 14 hrs. Also increased susceptibility to a bronchoconstrictor (acetylcholine).	
1,300 to 3,800	0.7 to 2.0	NO_2	10	10 mins.	Increased inspiratory and expiratory flow resistance of approximately 50% and 10% of control values measured 10 mins. after exposure.	Suzuki and Ishikawa, 1965
1,880	1.0	NO_2	16	2 hrs.	No statistically significant changes in pulmonary function tests with exception of small changes in FVC. (See page 1-9 and 15-17).)	Hackney et al. 1978
1,150	0.6	NO_2	15	2 hrs.	No physiologically significant changes in cardiovascular, metabolic, or pulmonary functions after 15, 30 or 60 mins. of exercise.	Folinsbee et al., 1978

TABLE 1-1. (continued)

Concentration μg/m³	 ppm	Pollu-tant	No. of Healthy Subjects	Exposure Time	Effects	Reference
1,000	0.50	O_3	4	4 hrs.	With each group minimal alterations in pulmonary function caused by O_3 exposure. Effects were not increased by addition of NO_2 or NO_2 and CO to test atmospheres.	Hackney et al., 1975
1,000 with	0.50	O_3				
560	0.29	NO_2				
1,000 with	0.50	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				
500	0.25	O_3	7	2 hrs.	Little or no change in pulmonary function found with O_3 alone. Addition of NO_2 or of NO_2 and CO did not noticeably increase the effect. Seven subjects included some believed to be unusually reactive to respiratory irritants.	Hackney et al., 1975
500 with	0.25	O_3				
560	0.29	NO_2				
500 with	0.25	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				
1,880 to 3,760	1.0 to 2.0	NO_2	10	2 1/2 hrs	Alternating exercise and rest produced significant decrease for hemoglobin, hematocrit, and erythrocyte acetylcholinesterase.	Posin et al., 1978

TABLE 1-1. (continued)

Concentration μg/m³	Concentration ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
100 with	0.05	NO_2	11	2 hrs.	No effect on R_{aw} or $AaDO_2$; exposed subjects showed increased sensitivity of bronchial tree to a bronchoconstrictor (acetylcholine) over controls not exposed to pollutants.	von Nieding et al., 1977
50 and	0.025	O_3				
300	0.11	SO_2				

*R_{aw} : airway resistance

$AaDO_2$: difference between alveolar and arterial blood partial pressure of oxygen

DL_{CO} : diffusion capacity of the lung for carbon monoxide

PaO_2 : arterial partial pressure of oxygen

PO_2 : partial pressure of oxygen

PCO_2 : partial pressure of carbon dioxide

**By descending order of lowest concentration evoking a significant effect.

***Reference citations are for studies listed in the bibliography for Chapter 15.

In a similar study, Kerr et al. (1979) exposed 10 normal healthy adults and 20 subjects with asthma and chronic bronchitis to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 2 hours. Although the authors suggest that the changes reported in quasistatic compliance for normal healthy adults may be due to chance alone, there is uncertainty whether these changes were due to normal daily variation or to NO_2 exposure. No other pulmonary function tests showed significant changes for the healthy adult group. Only one of the healthy adult group reported mild symptomatic effects associated with exposure to NO_2. The results of the Kerr study concerning asthmatics and chronic bronchitics are discussed later.

Beil and Ulmer (1976) and Folinsbee et al. (1978) concluded that there were no physiologically significant pulmonary effects at exposure levels of 1880 and 1100 $\mu g/m^3$ (1.0 and 0.6 ppm) NO_2, respectively. Hackney et al. (1975a,b,c) and von Nieding et al. (1977) also concluded that there were no physiologically significant effects at NO_2 levels below 560 $\mu g/m^3$ (0.3 ppm) in the presence of various other air pollutants, with the exception of increased sensitivity to a bronchoconstrictor (acetylcholine) observed by von Nieding et al. (1977) at 94 $\mu g/m^3$ (0.05 ppm) NO_2 in the presence of 49 $\mu g/m^3$ (0.025 ppm) ozone and 290 $\mu g/m^3$ (0.11 ppm) SO_2.

The latter von Nieding et al. (1977) finding, however, is difficult to interpret in view of: (1) controversy over the health significance of altered sensitivity to bronchoconstrictors in healthy subjects; (2) some uncertainties due to methodological differences between his techniques and other investigators'; and (3) no confirmation of the von Nieding et al. (1977) findings by other investigators. Though the von Nieding et al. (1977) findings are interesting, they cannot be accepted at this time as providing conclusive evidence for respiratory effects occurring at NO_2 concentrations substantially below 1880 $\mu g/m^3$ (1.0 ppm) for healthy adult subjects.

Several controlled clinical studies have also addressed the issue of whether detectable respiratory effects can be induced by NO_2 in sensitive human subjects at exposure levels below those affecting healthy human adults. Key clinical studies of the effects of exposure to NO_2 on pulmonary function in potentially susceptible groups of the population are presented in Table 1-2.

The studies by von Nieding et al. (1971; 1973) show that, in persons with chronic bronchitis, concentrations of 7,500 and 9,400 $\mu g/m^3$ (4.0 and 5.0 ppm) produced decreases in arterial partial pressure of oxygen and increases in the difference between alveolar and arterial partial pressure of oxygen. Exposures to concentrations of NO_2 above 2,800 $\mu g/m^3$ (1.5 ppm), for periods considerably less than 1 hour, also produced significant increases in airway resistance. Thus, results for bronchitic individuals and healthy individuals appear to differ little.

In contrast to the above results for bronchitics, exposures to 190 $\mu g/m^3$ (0.1 ppm) NO_2 for 1 hour were reported by Orehek (1976) to increase mean airway resistance (R_{aw}) in 3 of 20 asthmatics and to increase the sensitivity to a bronchoconstrictor (carbachol) in 13 of 20 of the same individuals. In another study (Kerr et al., 1979), however, measurements of pulmonary

TABLE 1-2. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION IN CONTROLLED STUDIES OF SENSITIVE HUMAN ADULTS

Concentration		No. of Subjects	Exposure Time	Effects*	Reference
$\mu g/m^3$	ppm				
9,400	5.0	14 chronic bronchitics	60 mins.	No change in mean PAO_2, during or after exposure compared with pre-exposure values, but PaO_2 decreased significantly in the first 15 mins. Continued exposure for 60 mins. produced no enhancement of effect.	von Nieding et al, 1973
3,800 to 9,400	2.0 to 5.0	25 chronic bronchitics	10 mins.	Significant decrease in PaO_2 and increase in $AaDO_2$ at 7,500 $\mu g/m^3$ (4.0 ppm) and above; no significant change at 3,800 $\mu g/m^3$ (2.0 ppm).	von Nieding et al., 1971
940 to 9,400	0.5 to 5.0	63 chronic bronchitics	30 inhalations	Significant increase in R_{aw} above 3,000 $\mu gm/^3$ (1.6 ppm); no significant effect below 2,800 $\mu g/m^3$ (1.5 ppm).	von Nieding et al., 1971
940	0.5	10 healthy 7 chronic bronchitics 13 asthmatics	2 hrs.	1 healthy and 1 bronchitic subject reported slight nasal discharge. 7 asthmatics reported slight discomfort. Bronchitics and asthmatics showed no statistically significant changes in any pulmonary functions tested when analyzed as separate groups but showed small, statistically significant changes in quasistatic compliance when analyzed as a single group.	Kerr, et al., 1979
190	0.1	20 asthmatics	1 hr.	Significant increase in SR_{aw} in 3 of 20 asthmatics. Effect of bronchoconstriction due to carbachol enhanced after exposure in 13 of 20 asthmatic subjects. Neither effect observed in 7 of 20 subjects.	Orehek, 1976

*PAO_2 : alveolar partial pressure of oxygen

R_{aw} : airway resistance

SR_{aw} : specific airway resistance

$AaDO_2$: difference between alveolar and arterial blood partial pressure of oxygen

PaO_2 : arterial partial pressure of oxygen function were not altered in 13 asthmatics or 7 bronchitics as a result of 2 hours of exposure to 940 μg/m^3 (0.5 ppm) NO_2 when the groups were analyzed separately. When the data for the two groups were analyzed together, small but statistically significant changes in quasistatic compliance and functional residual capacity were reported. However, the authors state that the changes reported may be due to chance alone. Seven asthmatics and one bronchitic reported some chest discomfort, dyspnea, headache, and/or slight nasal discharge.

It should be noted that considerable controversy exists regarding interpretation of the Orehek (1976) study and the health significance of the increased response to a bronchoconstrictor observed in the study. Conclusive statements regarding the possible unique status of asthmatics therefore, cannot be made at this time. If the Orehek and Kerr studies are further corroborated, however, then it is suggested that impairment of lung function in asthmatics may be affected at NO_2 levels of 940 μg/m^3 (0.5 ppm) or below.

The above controlled human exposure studies provide important data concerning the effects of single short-term NO_2 exposures on healthy young adults and certain groups defined a priori as "sensitive", i.e., bronchitics and asthmatics. However, members of other presumed sensitive populations, e.g., children, the elderly, and individuals with chronic cardiovascular disease, have not been tested in controlled exposure studies and are not likely to be tested in the future. Because of constraints on the study of such sensitive polulation groups in controlled exposure experiments, the question of whether such individuals are at greater risk than healthy young adults for experiencing respiratory effects with single or repeated short-term NO_2 exposures cannot be conclusively answered, although existing epidemiological data suggest that this may be the case for young children.

Other critical health effects issues cannot be adequately addressed by controlled clinical studies. These include the issues of: (1) assessment of pulmonary impairments induced by repeated short-term peak exposures or continuous low-level exposures to NO_2; (2) assessment of possible exacerbation of other disease processes by such NO_2 exposures; and (3) evaluation of morphological or structural tissue damage associated with NO_2 exposures, whether of a short-term, repeated or continuous nature. Some information bearing on the above issues has been obtained by human epidemiological and animal toxicological studies summarized below.

1.3.1.2 Human Epidemiological Studies--Epidemiological studies of the effects of community air pollution are complicated because there are usually complex mixtures of pollutants in the air. Thus, the most that can typically be obtained from such studies is the demonstration of close associations between health effects and ambient concentrations of a given mixture of pollutants or subfractions of the mixtures. Furthermore, the association must remain consistent throughout a variety of conditions for likely causality to be ascribed to such observations and only if other possible confounding or covarying factors have been adequately taken into account. Epidemiological studies of air pollution effects are also often hampered by difficulty in defining actual exposures of study populations.

It is important to note that community epidemiological studies prior to 1973 on the effects of NO_2 exposure are of questionable validity due to the use of the Jacobs-Hochheiser technique in measuring atmospheric concentrations of NO_2.* For this reason, the contributions of those community studies to knowledge concerning the effects of NO_2 are very limited. Certain other community exposure studies, however, provide better aerometric data as bases for attempting to quantify ambient air NO_2 exposure effects and are the central focus of the present analysis.

Community exposure studies investigating NO_2 effects on pulmonary function and providing quantitative data on associated ambient air levels of NO_2 are summarized in Table 1-3. Most of these studies consistently tend to indicate that reported daily mean concentrations of peak levels of NO_2, or NO_2 in combination with other pollutants (all less than 1.0 ppm NO_2) typically had no significant effects on lung function in the exposed study populations.

An exception is the Kagawa and Toyama (1975) study which showed some correlations in 20 Japanese schoolchildren, 11 years of age, between decrements in maximum expiratory flow rate ($\dot{V}_{max}$) or specific airway conductance and concentrations of NO_2 or other specific pollutant levels at the time of testing. One-hr. NO_2 concentrations during testing ranged from 40 to 360 $\mu g/m^3$ (0.02 to 0.19 ppm), but the data were such as not to allow for quantitative estimation of specific NO_2 levels that might have been associated with the occurrence of pulmonary function decrements. Also in the ambient situation the effects observed in this study were generally not associated with NO_2 alone, but rather with various combinations of air pollutants, including SO_2, particulate matter, and O_3. In addition, weekly variations in specific airway conductance and in $\dot{V}_{max}$ at 25 percent FVC were most closely correlated with outdoor temperature levels. These results emphasize that the observed respiratory effects resulted from a complex interaction of pollutants including NO_2 and do not allow for clear attribution of an association of decreased lung function with any specific ambient air concentration of NO_2.

Linn et al. (1976), Cohen et al. (1972), Burgess et al. (1973), and Speizer and Ferris (1973a,b) found no differences in pulmonary function tests in separate epidemiological studies which also involved complex pollutant mixtures in ambient air.

Several of the above community epidemiological studies also evaluated relationships between ambient air exposures to NO_2 at levels reported in Table 1-3 and the occurence of chronic respiratory diseases, but found no significant associations between the ambient NO_2 exposures and the health endpoints measured. A few other community epidemiology studies have been published which report quantitative associations between ambient NO_2 exposures and increased acute respiratory disease incidence, but the methods employed in those studies (e.g. use of the Jacobs-Hochheiser method for monitoring NO_2 levels) were such so as to preclude acceptance of the reported quantitative findings.

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*The Jacobs-Hochheiser technique has been withdrawn by EPA and replaced by a new Federal Reference Method (chemiluminescence) and other equivalent methods (Chapter 7).

TABLE 1-3. QUANTITATIVE COMMUNITY HEALTH EPIDEMIOLOGICAL STUDIES ON EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION

Measure	NO_2 Exposure Concentrations μg/m^3	NO_2 Exposure Concentrations ppm	Study Population	Effect	Reference
High exposure group:					
Annual mean 24-hr concentrations	96	0.051	Nonsmokers Los Angeles (adult)	No differences in several ventilatory measurements including spirometry and flow volume curves	Cohen et al., 1972
90th percentile	188	0.01			
Estimated 1-hr maximum[a]	480 to 960	0.26 to 0.51			
Low exposure group:					
Annual mean 24-hr concentrations	43	0.01	Nonsmokers San Diego (adult)		
90th percentile	113	0.06			
Estimated 1-hr maximum[a]	205 to 430	0.12 to 0.23			
Mean "annual"[b] 24-hr concentrations: high exposure area	103 + 92 SO_2	0.055 + 0.035 SO_2	Pulmonary function tests administered to 128 traffic policemen in urban Boston and to 140 patrol officers in nearby suburban areas.	No difference in various pulmonary function tests.	Speizer and Ferris, 1973a,b Burgess et al., 1973
low exposure area	75 + 36 SO_2	0.04 + 0.014 SO_2			
1-hr mean: high exposure area	260 to 560	0.14 to 0.30			
low exposure area	110 to 170	0.06 to 0.09			

TABLE 1-3. (continued)

Measure	NO_2 Exposure Concentrations µg/m^3	ppm	Study Population	Effect	Reference
Los Angeles:					
Median hourly NO_2	130	0.07	205 office workers in Los Angeles	No differences in most tests. Smokers in both cities showed greater changes in pulmonary function than non-smokers.	Linn, et al., 1976
90th percentile NO_2	250	0.13			
Median hourly O_x		0.15			
90th percentile O_x		0.15			
San Francisco:					
Median hourly NO_2	65	0.35	439 office workers in San Francisco		
90th percentile NO_2	110	0.06			
Median hourly O_x		0.02			
90th percentile O_x		0.03			
1-hr concentration at time of testing (1:00 p.m.)	40 to 360	0.02 to 0.19	20 school children 11 years of age	During warmer part of the year (April-October) NO_2, SO_2 and TSP* significantly correlated with $\dot{V}_{max}$* at 25% and 50% FVC* and with specific airway conductance. Temperature was the factor most clearly correlated with weekly variations in specific airway conductance with $\dot{V}_{max}$ at 25% and 50% FVC. Significant correlation between each of four pollutants (NO_2, NO, SO_2 and TSP) and $\dot{V}_{max}$ at 25% and 50% FVC; but no clear delineation of specific pollutant concentrations at which effects occur.	Kagawa and Toyama, 1975

[a] Estimated at 5 to 10 times annual mean 24-hour averages

[b] Mean "annual" concentrations derived from 1-hour measurements using Saltzman technique

*$FEV_{0.75}$: Forced expiratory volume, 0.75 seconds

$\dot{V}_{max}$: Maximum expiratory flow rate

FVC : Forced vital capacity

TSP : Total suspended particulates

As for the results of other epidemiological studies, some support for accepting the hypothesis that children are at special risk for NO_2-induced increases in acute respiratory illnesses is derived from certain British and United States studies on indoor pollution effects as summarized in Table 1-4.

These studies investigated possible decrements in lung function and/or increased respiratory symptom and illness rates among children living in homes using gas stoves for cooking in comparison to children from homes with electric ranges. Such studies are pertinent for present purposes because high temperature gas combustion is a source of NO_2.

Several studies substantiate that higher NO_2 levels accumulate in homes using gas stoves in comparison to NO_2 levels found in homes with electric stoves. Melia et al. (1978) reported that average NO_2 concentrations in 2 homes over a 96-hour test period, during which stoves were in use for 8.5 to 10 hours, were 136 $\mu g/m^3$ (0.072 ppm) when gas was burned and 18 $\mu g/m^3$ (0.01 ppm) in 2 other homes where electricity was used. In this study the NO_2 concentrations were monitored at 1.2 meters (4 ft) above floor level and 0.6 and 2.2 meters (2 and 7.5 feet) from either gas or electric stoves. Other studies, including Goldstein et al. (1979) and Spengler et al. (1979), confirm that the levels of NO_2 in gas stove homes are higher than those in homes using electric stoves, and studies by Wade et al. (1975) and Mitchell et al. (1974) also provide additional estimates of indoor NO_2 levels resulting from gas stove usage. For example, Wade et al. (1975) reported recurrent daily levels which averaged 280 $\mu g/m^3$ (0.15 ppm) in the kitchen for two hours around the time when peak concentrations were generated. In addition to variations in cooking routine, it should be noted that a variety of factors would be expected to influence short-term peak averages including interior house design, type and adjustment of range burners, and presence or absence of positive ventilation. An instantaneous peak of 1,880 $\mu g/m^3$ (1.0 ppm) was also measured on one occasion. Long-term average NO_2 concentrations, over observation periods of up to 2 weeks ranged from 103 to 145 $\mu g/m^3$ (0.055 to 0.077 ppm).

In an initial publication regarding the British studies on health effects associated with gas stove usage, Melia et al. (1977) reported a weak association between increased respiratory illness in school children and residence in homes using gas stoves versus electric stoves, after a number of demographic and other potentially confounding variables were taken into account. However, Melia et al. (1977) failed to adjust for tobacco smoking in the home in their first analysis. In other later publications (Melia et al., 1979; Goldstein et al., 1979; Florey et al., 1979) corrections were made for the number of smokers in the home and, again, weak associations between gas cooking and respiratory illness in children were found (independent of smoking and other factors) in urban areas but not in rural ones. There appeared, however, to be an association in rural areas for girls under the age of eight. Additionally, four cohorts of children followed longitudinally from 1973 to 1977 initially

TABLE 1-4. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE IN THE HOME ON LUNG FUNCTION AND THE INCIDENCE OF ACUTE RESPIRATORY DISEASE IN EPIDEMIOLOGY STUDIES OF HOMES WITH GAS STOVES

Pollutant[a]	NO_2 Concentration $\mu g/m^3$ ppm	Study Population	Effects	Reference
Studies of Children				
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study	2554 children from homes using gas to cook compared to 3204 children from homes using electricity. Ages 6-11	Proportion of children with one or more respiratory symptoms or disease (bronchitis, day or night cough, morning cough, cold going to chest, wheeze, asthma) increased in homes with gas stoves vs. electric stove homes (for girls $p \simeq 0.10$; boys not sig.) after controlling for confounding factors.	Melia et al., 1977
NO_2 plus other gas stove combustion products	NO_2 concentration not measured in same homes studied	4827 children ages 5-10	Higher incidence of respiratory symptoms and disease associated with gas stoves (for boys $p \simeq 0.02$; girls $p \simeq 0.15$) after controlling for confounding factors	Melia et al., 1979
NO_2 plus other gas stove combustion products	Kitchens: 9-596 (gas) 0.005-0.317 11-353 (elec) 0.006-0.188 Bedrooms: 7.5-318 (gas) 0.004-0.169 6 - 70 (elec) 0.003-0.037 (by triethanolamine diffusion samplers)	808 6- and 7-year-old children	Higher incidence of respiratory illness in gas-stove homes ($p \simeq 0.10$). Prevalence not related to kitchen NO_2 levels, but increased with NO_2 levels in bed rooms of children in gas-stove homes. Lung function not related to NO_2 levels in kitchen or bedroom	Florey et al., 1979 Companion paper to Melia et al., 1979; Goldstein et al., 1979
NO_2 plus other gas stove combustion products	95 percentile of 24 hr avg in activity room 39 - 116 $\mu g/m^3$ (.02 - .06 ppm) (gas) vs. 17.6 - 95.2 $\mu g/m^3$ (.01 - .05 ppm) (electric). Frequent peaks $\simeq$ 1100 $\mu g/m^3$ (0.6 ppm), max peak ~ 1880 $\mu g/m^3$ (1.0 ppm) 24 - hr by modified sodium arsenite; peaks by chemiluminescence	8,120 children 6-10 yrs old in 6 different communities with data collected on lung function and history of illness before the age of 2	Significant association between history of serious respiratory illness before age 2 and use of gas stoves ($p < .01$) and, also, between lower FEV, FVC levels and use of gas stoves ($p < .01$)	Speizer et al., 1980 Spengler et al., 1979

TABLE 1-4 (continued)

Pollutant[a]	NO_2 Concentration $\mu g/m^3$ ppm	Study Population	Effects	Reference
NO_2 plus other gas stove combustion products	Sample of households 24 hr avg: gas (.005 - .11 ppm); electric (0 - .06 ppm); outdoors (.015 - .05 ppm); several peaks > 1880 $\mu g/m^3$ (1.0 ppm). Monitoring location not reported. 24-hr avgs by sodium arsenite; peaks by chemiluminescence	128 children 0-5 346 children 6-10 421 children 11-15	No significant difference in reported respiratory illness between homes with gas and electric stoves in children from birth to 12 years	Mitchell et al., 1974 See also Keller et al., 1979a
NO_2 plus other gas stove combustion products	Sample of same households as reported above but no new monitoring reported	174 children under 12	No evidence that cooking mode is associated with the incidence of acute respiratory illness	Keller et al., 1979b
Studies of Adults				
NO_2 plus other gas stove combustion products	Preliminary measurements peak hourly 470 - 940 $\mu g/m^3$ max 1880 $\mu g/m^3$ (1.0 ppm)	Housewives cooking with gas stoves, compared to those cooking with electric stoves	No consistent statistically significant increases in respiratory illness associated with gas stove usage	USEPA, 1976
NO_2 plus other gas stove combustion products	See table above for monitoring	Housewives cooking with gas stoves, compared to those cooking with electric stoves. 146 households	No evidence that cooking with gas associated with an increase in respiratory disease	Keller et al., 1979a
NO_2 plus other gas stove combustion products	See table above for monitoring	Members of 441 households	No significant difference in reported respiratory illness among adults in gas vs electric cooking homes	Mitchell et al., 1974 See also Keller et al., 1979a
NO_2 plus other gas stove combustion products	See table above for monitoring	Members of 120 households (subsample of 441 households above)	No significant difference among adults in acute respiratory disease incidence in gas vs electric cooking homes	Keller et al., 1979b

showed greater risk of having one or more respiratory symptoms or diseases in homes with gas stoves, but the strength of the association varied greatly over the four year study period and was non-significant for some subgroups. In reviewing their overall results, the British authors expressed concern that other, potentially confounding factors, such as temperature and humidity, may have contributed to the apparent relationship between gas stove usage and increased respiratory illness in school children. The same investigators found no relationship between gas stove usage and lung function levels in subsets of the same children.

Based on initial results from a continuing prospective epidemiological study in the United States, Speizer et al. (1980) reported that children from households with gas stoves had a greater history of serious respiratory illness before age 2. In this study, adjustment of rates of illness before age 2 for parental smoking, socioeconomic status, and other factors led to a clear association between increased respiratory illness and the presence of gas-cooking devices. Also found were small but statistically significant lower levels of two measures of pulmonary function (corrected for height) in school age children from houses with gas stoves. Monitoring of a subset of the homes with gas facilities revealed that NO_2 was present in much higher concentrations than in the outside air, as reported by Spengler et al. (1979). Continuous NO_2 measurements in a residence with gas stoves showed that levels exceeding 500 $\mu g/m^3$ and even 1000 $\mu g/m^3$ can occur during cooking, with such high levels lasting from minutes to hours. Kitchen annual means may exceed 100 $\mu g/m^3$ (0.6 ppm) if one extrapolates from other studies, as noted by Spengler et al. (1979). Further, short-term hourly NO_2 kitchen levels during cooking were noted as possibly being 5 to 10 times higher than measured mean values. This is in contrast to annual average NO_2 levels of about 0.02 ppm (and no marked peaks) in homes with electric stoves.

These findings were interpreted as suggesting that repeated peak short-term exposures to NO_2 may be associated with increased incidence of respiratory illness in young preschool-age children and small decrements in lung function in school age children. Hypothesizing of such effects being associated with repeated short-term peak NO_2 exposures is based on annual average levels of NO_2 not being very different in the gas stove homes versus electric stove homes studied by Spengler et al. (1979) and Speizer et al. (1980). However, more definitive documentation of such respiratory system effects being associated with short-term NO_2 peak exposures remains to be provided based on further data collected beyond those included in the initial analyses discussed by Speizer et al. (1980) and Spengler et al. (1979) from their continuing prospective epidemiological study. This includes confirmation of the basic findings reported, confirmation of the exclusion of socioeconomic status and other confounding factors as important contributors to the observed relationships, and more detailed monitoring of indoor NO_2 levels in homes of children in the study populations.

Further complicating interpretation of the above findings and determination of the strength and reliability of the apparent relationships suggested by them are other studies which failed to find any associations between gas stove usage and increased respiratory illness or decrements in lung function. In a series of three related studies, Mitchell et al.

(1974) and Keller et al. (1979a,b) report negative findings with respect to an association between the use of gas stoves in the home and an increased incidence of respiratory disease in both adults and children. Initially 441 households in a middle-class suburb in Ohio were studied. A follow-up study on a subsample of 120 of the original 441 households was also conducted to verify the methodology used previously to collect health data. In addition, these investigators studied a group of 146 housewives in a community on Long Island, N.Y., confirming the negative results reported earlier by the U.S. EPA (1976). However, the sample sizes for children used in these studies were approximately a factor of 10 smaller than those used in both the British and American studies reporting an association between increased respiratory disease and gas cooking. Also, it is not clear that the households sampled in Ohio constituted a representative sample of the community studied.

1.3.1.3 Animal Toxicology Studies--In addition to epidemiological studies, animal toxicology studies provide valuable information regarding induction of respiratory system effects by NO_2. Although it is recognized that exposure/effect relationships demonstrated by animal studies are generally not quantitatively directly extrapolatable as indicators of human health effects thresholds, they are, nevertheless, highly instructive regarding probable mechanisms by which NO_2 may affect human pulmonary function and damage the pulmonary defense system, possibly causing increased human susceptibility to bacterial infections. Rather than attempt an exhaustive summary here of the animal toxicology literature assessed in Chapter 14, the present section concisely highlights key points from that literature as they relate to effects observed in human studies.

The lowest concentration of NO_2 that has been shown to produce measurable responses in animals is 376 $\mu g/m^3$ (0.2 ppm). At this concentration, rats exposed for 3 hours had an inhibition of the lung metabolic conversion of prostaglandin E_2 (a mediator which acts on smooth muscle) to its inactive 15-keto metabolite 18 hours after exposure ceased. This could possibly lead to an alteration of vascular or airway smooth muscle tone and, therefore, may ultimately contribute to the mediation of pulmonary function changes, e.g., increased airway resistance, observed with human exposure to higher levels ($\geq$1.0 ppm) of NO_2. Since the prostaglandin system is intimately involved in the local regulation of blood flow in the lung, alterations in prostaglandin metabolism may also have profound effects on the perfusion of the lung and, subsequently, on the gas exchange of the affected lung.

Morphological alterations in animal lung tissue, ranging from very small changes in collagen to emphysema-like changes have also been observed following NO_2 exposure. Very small changes occurred in rabbits after 24 or 36 days of exposure (4 hr/day, 5 days/wk) to 470 $\mu g/m^3$ (0.25 ppm). The health significance of the small collagen changes at low exposure levels are unknown, but it should be noted that collagen metabolism is disrupted in man and animals during fibrosis. Thus the initial small collagen effects seen after low-level NO_2 exposure may be indicative of the initiation of processes of increasingly greater health significance as they intensify at high NO_2 exposure levels. Emphysema-like changes were found in several species, most often after chronic exposure to high concentrations (18,800 μg/m^3, 10 ppm) far above ambient air NO_2 levels.

Systemic effects have also been observed after NO_2 exposure. Female mice receiving 470 μg/m^3 (0.25 ppm) for 3 hours exhibited an increase in pentobarbital-induced sleeping time. Such a response implies a potential effect on some aspect(s) of liver xenobiotic metabolism. Hematological effects have also been observed in guinea pigs after 7 days of exposure to both 690 μg/m^3 (0.36 ppm) and 940 μg/m^3 (0.5 ppm). There is no evidence available establishing that the same NO_2 exposure concentrations may produce similar systemic effects in human beings, and the potential significance of such effects for human health is unclear at this time.

Of more obvious importance are the results of other extensive animal studies that appear to be consistent with emerging conclusions from human epidemiological studies on gas stove usage discussed earlier. The latter studies appear to suggest that increased incidence of acute respiratory illness in humans may occur as a result of repeated short-term exposures to NO_2. Pertinent animal studies summarized in Table 1-5 have demonstrated that repeated short-term exposures increase susceptibility to some respiratory pathogens as much as does continuous exposure to the same concentration of NO_2 (see Section 14.2.3.1.1). NO_2 exposures causing increased infectivity in animals have been observed across a wide range, beginning at 940 μg/m^3 (0.5 ppm) for repeated exposures during a 90-day period and 3760 μg/m^3 (2.0 ppm) for single (3 hr) exposures. The results are interpreted here as providing evidence supportive of tentative conclusions emerging from the above "gas stove" studies, i.e., that repeated exposure to daily peak concentrations of NO_2 may be effective in impairing the health of exposed young children by increasing vulnerability to infectious respiratory diseases. The latter conclusions from the gas stove studies, however, still must be viewed with caution until more definitive results are obtained. From the mouse studies, it is also apparent that concentration of NO_2 has more importance than time of exposure in producing increased susceptibility to bacterial infection. Of interest, the lowest NO_2 concentrations (0.5 to 1.5 ppm) found to induce such effects in animals with repeated or intermittent continuous exposures, do not greatly exceed to the upper range of repeated NO_2 peak levels found in gas stove usage homes hypothesized to be associated with increased respiratory illnesses in children.

1.3.2 NO_2 Sensory System Effects

In addition to the effects of NO_2 on pulmonary functions and its possible association with increased acute respiratory disease in young children, NO_2 also exerts discernible effects on sensory receptors (Table 1-6). This includes the detection of NO_2 as a noxious pungent odor starting at concentration levels as low as 210 μg/m^3 (0.11 ppm) of NO_2 and occurring immediately upon exposure. Under some exposure conditions, however, impairment of odor detection occurs. For example, impaired detection of NO_2 concentrations of 18,800 μg/m^3 (10 ppm) or more has been reported. In general, the former sensory effect, odor detection, is not viewed as a significant health effect of concern (although it could be construed as affecting human welfare); and the latter olfactory deficit effect, impairment of odor detection, is of likely negligible health concern in view of its temporary, reversible nature.

TABLE 1-5. SUMMARY OF STUDIES DEMONSTRATING HEALTH EFFECTS IN ANIMALS AT LOW (≤2.0 ppm) NO_2 EXPOSURE LEVELS

NO_2 Concentrations μg/m³	ppm	Effects	NO_2 measurement method	Reference
3,760	2.0	A single 3-hr exposure caused increased mortality following challenge with an infectious agent in mice.	Chemiluminescence	Ehrlich et al., 1977
2,800	1.5	After 1 wk continuous exposure, mice had a significantly greater increase in susceptibility to infectious pulmonary disease compared to intermittent exposure. After 2 wks, no differences between continuous and intermittent exposure modes occurred.	Saltzman and Chemiluminescence procedures	Gardner et al., 1979
1,880 (daily 2 hr spike) 188 (continuous)	1.0 0.1	Emphysematous alterations in mice after 6 mo exposure.	Saltzman[a]	Port et al., 1977
940	0.5	In mice challenged with bacteria, there was increased mortality following a 90-day continuous exposure or a 180 day intermittent exposure.	Saltzman[a]	Ehrlich and Henry, 1968

TABLE 1-5. (continued)

NO_2 Concentrations μg/m³	ppm	Effects	NO_2 measurement method	Reference
940	0.5	A 7-day intermittent exposure caused enzymatic alterations in lungs and blood of guinea pigs.	Saltzman[a]	Donovan et al., 1976
940	0.5	Intermittent exposure for 6 hr/day for up to 12 mo caused morphological changes in lung alveoli of mice.		Blair et al., 1969
940	0.5	Increased susceptibility to influenza infection in mice.	Saltzman[a]	Ito, 1971
750	0.4	Continuous exposure for one wk caused increase of protein in lavage fluid in vitamin C deficient guinea pigs.		Sherwin and Carlson, 1973.
1880 (max peak reported) 50-350* (2-hr avg)	1.0 0.03-0.19	Swollen collagen fibers after an intermittent 24 or 36 day exposure of rabbits		Buell, 1970
667 to 94	0.36 to 0.05	Hematological effects observed in guinea pigs after 7 days of exposure to 690 or 940 μg/m³ (0.36 or 0.5 ppm).		Donovan et al., 1976 Menzel et al., 1977 Mersch et al., 1973

TABLE 1-5. (continued)

NO_2 Concentrations				
µg/m³	ppm	Effects	NO_2 measurement method	Reference
470	0.25	Increased pentobarbital-induced sleeping time in female mice after a 3 hr exposure. No effects after 2 or 3 days.		Miller et al., 1980
376	0.2	Inhibition of metabolism of prostaglandin E_2 in rats to its inactive 15-keto metabolite.		Menzel, 1980

[a] Saltzman, 1954 (See Chapter 7.)

TABLE 1-6. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON SENSORY RECEPTORS IN CONTROLLED HUMAN STUDIES

NO_2 Concentrations		No. of Subjects	Time until effect	Effects	No. of Subjects Responding	Reference
µg/m³	ppm					
790	0.42	8	Immediate	Perception of odor of NO_2	8/8	Henschler et al., 1960
410	0.22	13	Immediate	Perception of odor of NO2	8/13	Ibid.
230	0.12	9	Immediate	Perception of odor of NO_2	3/9	Ibid.
230	0.12	14	Immediate	Perception of odor of NO_2	most	Shalamberidze,
200	0.11	28	Immediate	Perception of odor of NO_2	26/28	Feldman, 1974
0 to 51,000	0 to 27	6	54 minutes	No perception of odor of NO_2 when concentration was raised slowly from 0 to 51,000 µg/m³	0/6	Henschler et al., 1960
2,260	1.2	6	Immediate	Perception of odor improved when relative humidity was increased from 55% to 78%	6/6	Ibid.
140	0.07	4	5 and 25 minutes	Impairment of dark adaptation	4/4	Shalamberidze,
150 to 500	0.08 to 0.26	5	Initial	Increased time for dark adaptation at 500 µg/m³ (0.26 ppm)	Not Reported	Bondareva, 1963
			Repeated over 3 months	Initial effect reversed		

NO_2 exposures also exert effects on other sensory perception functions. Probably most significant is the NO_2 effect on dark adaptation. Two different studies (Shalamberidze, 1976, and Bondareva, 1973) report data indicating that impairment of dark adaptation can occur in human subjects at NO_2 exposure levels as low as 130 to 150 $\mu g/m^3$ (.07 to .08 ppm). It is difficult to fully appraise the health significance of such an effect, but it appears to be of generally negligible concern except, perhaps, for certain occupational or public safety situations where rapid dark adaptation may be important.

1.3.3 Summary of Major Health Effects Conclusions

Major conclusions regarding NO_x-associated health effects of most importance for consideration in decision-making regarding primary National Ambient Air Quality Standards for NO_x compounds can be summarized as follows:

(1) At concentrations of 9,400 $\mu g/m^3$ (5.0 ppm) or above, exposure to NO_2 for as little as 15 minutes will both increase airway resistance in healthy human adults and impair the normal transport of gases between the blood and the lungs.

(2) In healthy adult individuals, concentrations of 4,700 $\mu g/m^3$ (2.5 ppm) NO_2 for 2 hours have been reported to increase airway resistance significantly without altering arterialized oxygen pressure. Single exposures for 15 minutes to NO_2 at concentrations of 3,000 $\mu g/m^3$ (1.6 ppm) are also likely to increase airway resistance in healthy adults and individuals with chronic bronchitis but not to interfere with the transport of gases between blood and lungs.

(3) Single exposures for times ranging from 15 minutes to 2 hours to NO_2 at concentrations of 2,800 $\mu g/m^3$ (1.5 ppm) or below have not been shown to affect respiratory function in healthy individuals or in those with bronchitis.

(4) Whether asthmatic subjects are more sensitive than healthy adults in experiencing NO_2-induced pulmonary function changes remains to be definitively resolved. The results of one controlled human exposure study suggest that some asthmatics may experience chest discomfort, dyspnea, headache, and/or slight nasal discharge following 2 hr exposures to 0.5 ppm NO_2 but did not provide convincing evidence of pulmonary function changes in asthmatics at that NO_2 concentration.

(5) Certain animal studies demonstrate various mechanisms of action by which pulmonary function changes of the above type may be induced in humans at relatively low NO_2 exposure levels and by which increasing by more serious histopathological changes leading to severe emphysematous effects are manifested at increasingly higher (generally greater than ambient) NO_2 exposure levels.

(6) Prospective studies of the effects of indoor air pollution suggest that, in some instances, an increased incidence of respiratory illness in young children may be associated with the use of gas stoves and possibly with NO_2 produced by these appliances. Much caution must be applied, however, in fully accepting or using these study findings for risk assessment purposes until: they are confirmed by further analyses of data subsequently gathered in the prospective studies; potential confounding factors are more definitively ruled out; and clearer exposure/effect relationships are defined via more intensive NO_2 monitoring in gas stove homes.

(7) No definitive estimates can yet be provided for peak 1-2 hr, 24 hr, weekly, or annual average NO_2 exposure levels that may be associated with any increased respiratory illness in young children residing in homes using gas stoves, although some basis exists for suggesting that repeated exposures to peak levels are most likely to be importantly involved. Peak 1-2 hr NO_2 levels ranging up to 0.5-1.0 ppm have been observed in gas stove homes; longer-term weekly average NO_2 levels of 0.05 to 0.07 ppm and annual average levels of 0.01 to 0.06 ppm were also found in such homes.

(8) Estimates of repeated short-term peak concentrations of NO_2 possibly associated with increased respiratory illness in homes with gas stoves are not markedly below the general range of the lowest (0.5 to 1.0 ppm) intermittent exposure concentrations found to cause increased susceptibility to respiratory infections in animal infectivity model studies.

Placing the above conclusions in perspective, it should be noted that ambient air NO_2 monitoring results in the United States indicate that peak 1-hr NO_2 concentrations rarely exceed 0.4 to 0.5 ppm. Such peaks occurred during 1975 to 1980 in only a few scattered locations in the United States, e.g., Los Angeles and several other California sites. Also, during that period, annual average NO_2 concentrations exceeding 0.05 ppm were only found in a relatively few scattered locations, including population centers such as Chicago and Southern California.

1.4 WELFARE EFFECTS OF NITROGEN OXIDES

In addition to human health effects associated with exposures to nitrogen dioxide discussed above, considerable attention has been accorded to the investigation of possible effects of NO_2 and other NO_x compounds on aquatic and terrestial ecosystems, vegetation, visibility, climate, and man-made materials--effects which may impact negatively on public welfare in the United States. Concisely summarized below are key points derived from more detailed discussions in Chapters 10-13 of the relationship of NO_x compounds to the induction of such welfare effects.

1.4.1 Nitrogen Oxides, Acidic Deposition Processes, and Effects

The occurrence of acidic deposition, especially in the form of acidic precipitation, has become a matter of concern in many regions of the United States, Canada, northern Europe, Taiwan and Japan. Acidic precipitation in the Adirondack Mountains of New York State, in Maine, in eastern Canada, in southern Norway and in southwest Sweden has been reported to be associated with acidification of waters in ponds, lakes and streams with a resultant disappearance of animal and plant life. Acidic precipitation (rain and snow) is also believed to have the potential to: (1) leach nutrient elements from sensitive soils, (2) cause direct and indirect injury to forests, (3) damage monuments and buildings made of stone, and (4) corrode metals.

Chapter 11 of this document discusses acidic deposition processes and the effects of wet deposition of sulfur and nitrogen oxides and their products on aquatic and terrestrial ecosystems. Dry deposition also plays an important role, but contributions by this process have not been well quantified. Because sulfur and nitrogen oxides are so closely linked in the formation of acidic precipitation, no attempt has been made to limit the present discussion solely to nitrogen oxides. A more thorough general review of acidic deposition processes and associated environmental problems will be presented in a future EPA document.

Sulfur and nitrogen oxides are considered to be the main precursors in the formation of acidic precipitation. Emissions of such compounds involved in acidification are attributed chiefly to the combustion of fossil fuels such as coal and oil. Emissions may occur at ground level, as from automobile exhausts, or from 300 meters (1000 feet) or more in height. Emissions from natural sources are also involved; however, in highly industrialized areas, emissions from manmade sources markedly exceed those from natural sources. In the eastern United States the highest emissions of sulfur oxides derive from electric power generators burning coal. However, emissions of nitrogen oxides, mainly from automotive sources, tend to predominate in the West. (Information regarding sources and emissions of NO_x compounds is discussed in Chapter 5 of this document.)

The fate of sulfur and nitrogen oxides, as well as other pollutants emitted into the atmosphere, depends on their dispersion, transport, transformation and deposition. Sulfur and nitrogen oxides or their transformation products may be deposited locally or transported long distances from the emission sources (Altshuller and McBean, 1976; Pack, 1978; Cogbill and Likens, 1974). Residence time in the atmosphere, therefore, can be brief if the emissions are deposited locally or may extend to days or even weeks if long range transport occurs. The chemical form in which emissions ultimately reach the receptor, the biological organism or material affected, is determined by complex chemical transformations that take place between the emission sources and the receptor. Long range transport over distances of hundreds or thousands of miles allows time for many chemical transformations to occur.

Sulfates and nitrates are among the products of the chemical transformations of sulfur oxides (especially SO_2) and nitrogen oxides. Ozone and other photochemical oxidants are believed to be involved in the chemical processes that form sulfates and nitrates. When sulfates and nitrates combine with atmospheric water, dissociated forms of sulfuric (H_2SO_4) and nitric (HNO_3) acids result; and when these acids are brought to earth in rain and snow, acidic precipitation occurs. Because of long range transport, acidic precipitation in a particular state or region can be the result of emissions from sources in states or regions many miles away, rather than from local sources. To date, however, the complex nature of the chemical transformation processes has not made it possible to demonstrate a direct cause and effect relationship between emissions of sulfur and nitrogen oxides and the acidity of precipitation. (Transport, transformation, and deposition of nitrogen compounds are discussed in Chapter 6 of this document; analogous information on sulfur oxides is discussed in a separate document, Air Quality Criteria for Particulate Matter and Sulfur Oxides, U.S. EPA, 1981).

Acidic precipitation has been arbitrarily defined as precipitation with a pH less than 5.6, because precipitation formed in a geochemically clean environment would have a pH of approximately 5.6 due to the combining of carbon dioxide with moisture in the air to form carbonic acid. Currently the acidity of precipitation in the northeastern United States usually ranges from pH 3.9 to 5.0; in other regions of the United States precipitation episodes with a pH as low as 3.9 have also been reported in areas with average pH levels above 5.0 (see Figure 1-1).

The pH of precipitation can vary from event to event, from season to season and from geographical area to geographical area. Other substances in the atmosphere besides sulfur and nitrogen oxides can cause the pH to shift by making it more acidic or more basic. For example, dust and debris swept up in small amounts from the ground into the atmosphere may become components of precipitation. In the West and Midwest soil particles tend to be more basic, but in the eastern United States they tend to be acidic. Also, in coastal areas sea spray strongly influences precipitation chemistry by contributing calcium, potassium, chlorine and sulfates. In the final analysis, the pH of precipitation is a measure of the relative contributions of all of these components (Whelpdale, 1978).

It is not presently clear as to when precipitation in the U.S. began to become markedly more acidic than the 5.6 pH level expected for a geochemically clean environment. Some scientists argue that it began with the industrial revolution and the burning of large amounts of coal and others estimate that it began in the United States with the introduction of tall stacks in power plants in the 1950's. However, other scientists disagree completely and argue that rain has always been acidic. In other words, no definitive answer to the question exists at the present time. Also, insufficient data presently exist to characterize with confidence long-term temporal trends in changes in the pH of precipitation in the United States, mainly due to the pH of rain not having been continuously monitored over extended periods of time.

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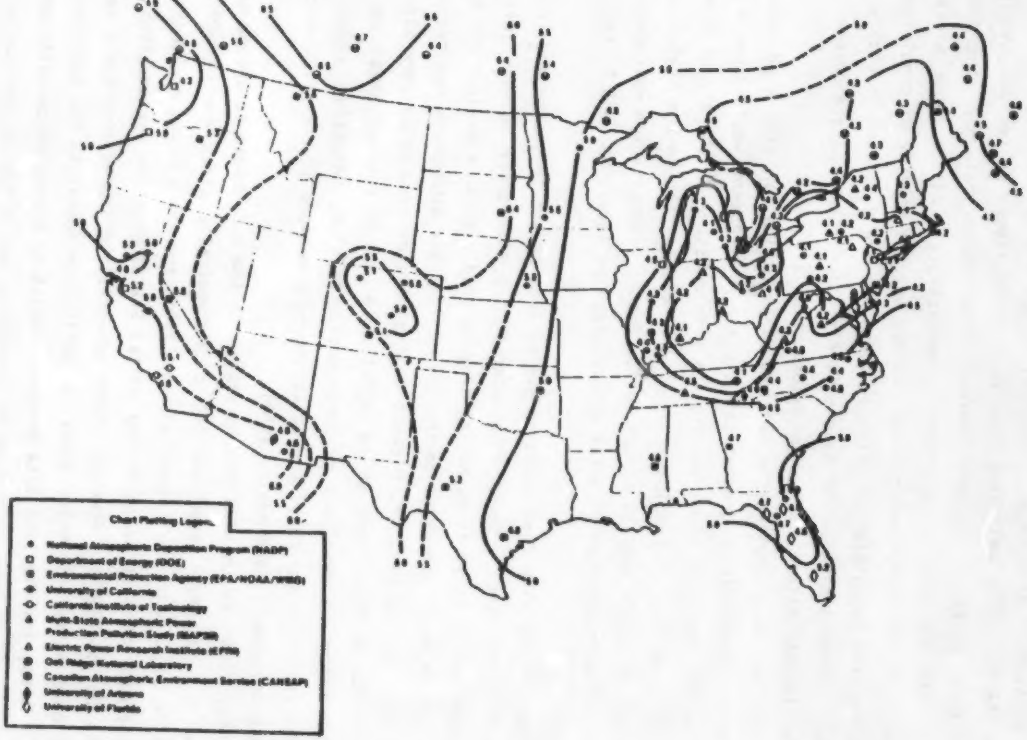
Chart Plotting Legend
● National Atmospheric Deposition Program (NADP)
□ Department of Energy (DOE)
▣ Environmental Protection Agency (EPA/NOAA/WMO)
◆ University of California
◇ California Institute of Technology
▲ Multi-State Atmospheric Power Production Pollution Study (MAP3S)
△ Electric Power Research Institute (EPRI)
● Oak Ridge National Laboratory
● Canadian Atmospheric Environment Service (CANSAP)
University of Arizona
University of Florida

Figure 1-1. Average pH isopleths as determined from laboratory analyses of precipitation samples, weighted by the reported quantity of precipitation.

Source: Wisniewski and Keitz (1981).

Although acidic precipitation (wet deposition) is usually emphasized, it is not the only process by which acids or acidifying substances are added to bodies of water or to the land. Dry deposition also occurs. Dry deposition processes include gravitational sedimentation of particles, impaction of aerosols and the adsorption and absorption of gases by objects at the earth's surface or by the soil or water. Dew, fog, and frost are also involved in the deposition processes but do not strictly fall into the category of wet or dry deposition (Galloway and Whelpdale, 1980; Sehmel, 1980; Hicks and Wesley, 1980). Dry deposition processes are not as well understood as wet deposition at the present time; however, all of the deposition processes contribute to the gradual accumulation of acidic or acidifying substances in the environment.

The most visible changes associated with acidic deposition, that is both wet and dry processes, are those observed in the lakes and streams of the Adirondack Mountains in New York State, in Maine, in the Pre-cambrian Shield areas of Canada, in Scotland, and in the Scandinavian countries. In these regions, the pH of the fresh water bodies appears to have decreased, causing changes in animal and plant populations.

The chemistry of fresh waters is determined primarily by the geological structure (soil system and bedrock) of the lake or stream catchment basin, by the ground cover and by land use. Near coastal areas (up to 100 miles inland) marine salts also may be important in determining the chemical composition of the stream, river or lake. Sensitivity of a lake to acidification depends on the acidity of both wet and dry deposition plus the same factors--the soil system of the drainage basin, the canopy effects of the ground cover and the composition of the waterbed bedrock. The capability, however, of a lake and its drainage basin to neutralize incoming acidic substances is determined largely by the composition of the bedrocks (Wright and Gjessing, 1976; Galloway and Cowling, 1978; Hendrey et al., 1980). Soft water lakes, those most sensitive to additions of acidic substances, are usually found in areas with igneous bedrock which contributes few soluble solids to the surface waters, whereas hard waters contain large concentrations of alkaline earths (chiefly bicarbonates of calcium and sometimes magnesium) derived from limestones and calcareous sandstones in the drainage basin. Alkalinity is associated with the increased capacity of lakes to neutralize or buffer the incoming acids. The extent to which acidic precipitation contributes to the acidification process has yet to be determined.

The survival of natural living ecosystems in response to marked environmental changes or perturbations depends upon the ability of constituent organisms of which they are composed to cope with the perturbations and to continue reproduction of their species. Those species of organisms most sensitive to particular environmental changes are first removed. However, the capacity of an ecosystem to maintain internal stability is determined by the ability of all individual organisms to adjust and survive, and other species or components may subsequently be impacted in response to the loss of the most susceptible species.

The capacity of organisms to withstand injury from weather extremes, pesticides, acidic deposition or polluted air follows the principle of limiting factors (Billings, 1978; Odum, 1971; Moran et al., 1980; Smith, 1980). According to this principle, for each physical factor in the environment there exists for each organism a minimum and a maximum limit beyond which no members of a particular species can survive. Either too much or too little of a factor such as heat, light, water, or minerals (even though they are necessary for life) can jeopardize the survival of an individual and in extreme cases a species. The range of tolerance (see Figure 1-2) of an organism may be broad for one factor and narrow for another. The tolerance limit for each species is determined by its genetic makeup and varies from species to species for the same reason. The range of tolerance also varies depending on the age, stage of growth or growth form of an organism. Limiting factors are, therefore, factors which, when scarce or overabundant, limit the growth, reproduction and/or distribution of an organism.

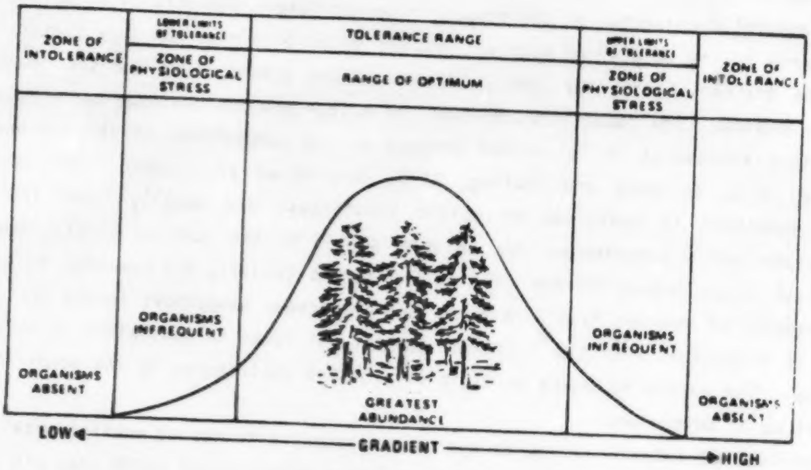
ZONE OF INTOLERANCE
LOWER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
TOLERANCE RANGE
RANGE OF OPTIMUM
UPPER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
ZONE OF INTOLERANCE
ORGANISMS ABSENT
ORGANISMS INFREQUENT
GREATEST ABUNDANCE
ORGANISMS INFREQUENT
ORGANISMS ABSENT
LOW
GRADIENT
HIGH

Figure 1-2. Idealized conceptual framework illustrating the "law of tolerance," which postulates a limited tolerance range for various environmental factors within which species can survive (adapted from Smith, 1980).

Continued or severe perturbation of an ecosystem can overcome its resistance or prevent its recovery, with the result that the original ecosystem will be replaced by a new system. In the Adirondack Mountains of New York State, in eastern Canada, and parts of Scandinavia the original aquatic ecosystems have been and are continuing to be replaced by ecosystems different from the original due to acidification of the aquatic habitat. Forest ecosystems, however, appear thus far to have been resistant to changes due to perturbation or stress from acidifying substances.

The impact of acidic precipitation on aquatic and terrestrial ecosystems is typically not the result of a single or several individual precipitation events, but rather the result of continued additions of acids or acidifying substances over time. Wet deposition of acidic substances on freshwater lakes, streams, and natural land areas is only part of the problem. Acidic substances exist in gases, aerosols, and particulate matter transferred into the lakes, streams, and land areas by dry deposition as well. Therefore, all the observed biological effects should not be attributed to acidic precipitation alone.

The disappearance of fish populations from freshwater lakes and streams is usually one of the most readily observable signs of lake acidification. Death of fish in acidified waters has been attributed to the modification of a number of physiological processes by a change in pH. Two patterns related to pH change have been observed. The first involves a sudden short-term drop in pH and the second, a gradual decrease in pH with time. Sudden short-term drops in pH may result from a winter thaw or the melting of the snow pack in early spring and the release of the acidic constituents of the snow into the water.

Long-term gradual increases in acidity, particularly below pH 5, interfere with reproduction and spawning, producing a decrease in population density and a shift in size and age of the population to one consisting primarily of larger and older fish. Effects on yield often are not recognizable until the population is close to extinction; this is particularly true for late-maturing species with long lives. Even relatively small increases (5 to 50 percent) in mortality of fish eggs and fry can decrease yield and bring about extinction.

In some lakes, concentrations of aluminum may be as crucial or more important than pH levels as factors causing a decline in fish populations in acidified lakes. Mobilization of certain aluminum compounds in the water due to lowered pH, upsets the osmoregulatory function of blood in fish. Aluminum toxicity to aquatic biota other than fish has not been assessed.

Although the disappearance of and/or reductions in fish populations are usually emphasized as significant results of lake and stream acidification, also important are the effects on other aquatic organisms ranging from waterfowl to bacteria. Organisms at all trophic (feeding) levels in the food web appear to be affected. Species reduction in number and diversity may occur, biomass (total mass of living organisms in a given volume of water) may be altered and processes such as primary production and decomposition impaired.

Significant changes that have occurred in aquatic ecosystems with increasing acidity include the following:

1. Fish populations are reduced or eliminated.
2. Bacterial decomposition is reduced and fungi may dominate saprotrophic communities. Organic debris accumulates rapidly, tying up nutrients, and limiting nutrient mineralization and cycling.
3. Species diversity and total numbers of species of aquatic plants and animals are reduced. Acid-tolerant species predominate.
4. Phytoplankton productivity may be reduced due to changes in nutrient cycling and nutrient limitations.
5. Biomass and total productivity of benthic macrophytes and algae may increase due in part to increased lake transparency.
6. Numbers and biomass of herbivorous invertebrates decline. Tolerant invertebrate species, e.g., air-breathing insects may become abundant primarily due to reduced fish predation.
7. Changes in community structure occur at all trophic levels.

An indirect effect of acidification potentially of concern to human health is the possible contamination of edible fish and of water supplies. Studies in Canada and Sweden reveal high mercury concentrations in fish from acidified regions. Lead has been found in plumbing systems with acidified water, and persons drinking the water could be affected by the lead. However, no examples have yet been documented of such human effects having actually occurred in response to acidic precipitation processes.

Soils may become gradually acidified from an influx of hydrogen (H^+) ions. Leaching of the mobilizeble forms of mineral nutrients may occur. The rate of leaching is determined by the buffering capacity of the soil and the amount and composition of precipitation. Unless the buffering capacity of the soil is strong and/or the salt content of precipitation is high, leaching will in time result in acidification. Anion mobility is also an important factor in the leaching of soil nutrients. Cations cannot leach without the associated anions also leaching. The capacity of soils to adsorb and retain anions increases as the pH decreases, when hydrated oxides of iron and aluminum are present (Wiklander, 1980).

Sulfur and nitrogen are essential for optimal plant growth. Plants usually obtain nitrogen in the form of nitrate from organic matter during microbial decomposition. Wet and dry deposition of atmospheric nitrates is also a major source. In soils where sulfur and nitrogen are limiting nutrients, such deposition may increase growth of some plant species. The amounts of nitrogen entering the soil system from atmospheric sources is dependent on proximity to industrial areas, the sea coast, and marshlands. The prevailing winds and the amount of precipitation in a given region are also important (Halstead and Rennie, 1977).

At present there are no documented observations or measurements of changes in natural terrestrial ecosystems or agricultural productivity directly attributable to acidic precipitation. The information available regarding vegetational effects concerns the results of a variety of controlled research studies, mainly using some form of "simulated" acidic rain, frequently dilute sulfuric acid. The simulated "acid rains" have deposited hydrogen (H^+), sulfate ($SO_4^=$) and nitrate (NO_3^-) ions on vegetation and have caused necrotic lesions in a wide variety of plants species under greenhouse and laboratory conditions. Such results must be interpreted with caution, however, because growth and morphology of leaves under such conditions are not necessarily typical of field conditions.

Damage to monuments and buildings made of stone, corrosion of metals and deterioration of paint may also result from acidic precipitation. Because sulfur compounds are a dominant component of acidic precipitation and are deposited during dry deposition as well, the effects resulting from the two processes cannot be clearly distinguished. Also, deposition of sulfur compounds on stone surfaces may provide a medium for microbial growth that can result in deterioration.

Several aspects of the acidic precipitation problem remain subject to debate because existing data are ambiguous or inadequate. Important unresolved issues include: (1) the rate at which rainfall is becoming more acidic and/or the rate at which the problem is becoming geographically more widespread; (2) the quantitative contributions of various acids to the overall acidity of rainfall; (3) the relative extent to which the acidity of rainfall in a region depends on local emissions of nitrogen and sulfur oxides versus emissions transported from distant sources; (4) the relative importance of changes in total mass emission rates compared to changes in the nature of the emission patterns (ground level versus tall stacks) in contributing to regional acidification of precipitation; (5) the relative contribution of wet and dry deposition to the acidification of lakes and streams; (6) the geographic distribution of natural sources of NO_x, SO_x and NH_3 and the significance and seasonality of their contributions; (7) the existence and significance of anthropogenic, non-combustion sources of SO_x, NO_x and HCl; (8) the dry deposition rates for SO_x, NO_x, sulfate, nitrate and HCl over various terrains and seasons of the year; (9) the existence and reliability of long-term pH measurements of lakes and headwater streams; (10) the acceptability of current models for predicting long range transport of SO_x and NO_x and for acid tolerance of lakes; (11) the feasibility of using liming or other corrective procedures to prevent or reverse acid damage and the costs of such procedures; (12) the effects of SO_x and NO_x and hydrogen ion deposition on ecosystem dynamics in both aquatic and terrestrial ecosystems; (13) the effectiveness of fertilization resulting from sulfate and nitrate deposition on soils; (14) the effects, if any, of acidic deposition on agricultural crops, forests and other native plants; and (15) the effects of acidic deposition on soil microbial processes and nutrient cycling. A more comprehensive evaluation of scientific evidence bearing on these issues is being prepared as part of a forthcoming EPA document on acidic deposition.

1.4.2 Effects of NO_x on Ecosystems and Vegetation

Chapter 12 discusses NO_x effects on ecosystems and vegetation. Ecosystems represent the natural order by which living organisms are bound to each other and to their environment. They are, therefore, essential to the existence of any species on earth, including man, and as life systems their value cannot be fully quantified in economic terms.

Ecosystems are important in the production of food, in the regeneration of essential nutrients as well as atmospheric components, in the assimilation or breakdown of many pollutants from the air, water, and soil, and in energy flow. They also give aesthetic pleasure and improve the quality of life.

The nitrogen cycle, an ecosystem function, is essential for all life because nitrogen is necessary in the formation of all living matter. Man has influenced the cycling of nitrogen by injecting fixed nitrogen into the environment or contributing other nitrogenous compounds which perturb the cycle.

Human activities have unquestionably increased the amounts of nitrates and related compounds in some compartments of the environment. The effects of such increased concentrations of nitrogen compounds may be beneficial or adverse, or both. Effects of both kinds may occur simultaneously, and may be felt in media or in ecological compartments quite removed from those that initially receive anthropogenic nitrogenous inputs.

Assessment of the influence of nitrogen oxides on ecosystems is complicated by several factors. Nitrogen oxides: (1) react with abiotic components of the natural environment as well as with individual organisms; (2) react with varying numbers of dissimilar populations within ecosystems; and (3) may suppress individual populations and thus affect ecosystem functioning.

One function of ecosystems is the cycling of nutrients such as nitrogen. Any effect, environmental or biological, which interferes with the recycling process could have a deleterious effect on the total ecosystem.

At the present time there are insufficient data to determine the impact of nitrogen oxides as well as other nitrogen compounds on terrestrial plant, animal or microbial communities. It is possible, however, to estimate the approximate magnitude of anthropogenic nitrogen fluxes to ecosystems, using the limited amount of monitoring data available or mass balance calculations. Such estimates, and quantitative information about the nitrogen cycle at specific sites in the system under study, make it possible to reach some conclusions about the possible ecological significance of the added nitrogen. In addition, where the data base is more extensive, as it is for a number of lakes in various stages of eutrophication, more quantitative dose-response relationships can be estimated.

A reduction in diversity within a plant community results in a reduction in the amount of nutrients present so that the growth of remaining individuals decreases.

Pollutants also act as predisposing agents so that disease, insect pests and abiotic forces can more readily injure the individual members of ecosystems. The loss of these individuals result in reduction in diversity and simplification of an ecosystem.

Sensitivity of plants to nitrogen oxides depends on a variety of factors including species, time of day, light, stage of maturity, type of injury examined, soil moisture, nitrogen nutrition and the presence or absence of other air pollutants such as sulfur dioxide and ozone.

When exposures to NO_2 alone are considered, the ambient concentrations that produce measurable injury are higher than those that normally occur in the United States (Chapter 8). Tomato (*Lycopersicon esculentum*) plants exposed continuously to 470 $\mu g/m^3$ (0.25 ppm) for 128 days exhibited a decreased growth and a yield reduction of 12 percent. Leaf drop and reduced yield occurred in orange trees exposed to 470 $\mu g/m^3$ (0.25 ppm) continuously for 8 months. Pinto bean (*Phaseolus vulgaris*), endive (*Cicorium endivia*) and cotton (*Gossypium hirsutum*) exhibited slight leaf spotting after 48 hours of exposure to 1,880 $\mu g/m^3$ (1.0 ppm). Other reports cited no injury in beans (*Phaseolus vulgaris*), tobacco (*Nicotiana tabacum*), or petunia (*Petunia multiflora*) with a 2-hour exposure to the same concentration.

Exceptions to this generality, however, have been observed. For example, the growth of Kentucky bluegrass was significantly reduced (approximately 25 percent) by exposures to 210 $\mu g/m^3$ (0.11 ppm) NO_2 for 103.5 hours per week for 20 weeks during the winter months. Similar exposures to other grass species generally had no deletrious effect on plant growth.

Nitrogen dioxide concentrations ranging from 188 to 1,880 $\mu g/m^3$ (0.1 to 1.0 ppm) increased chlorophyll content in pea (*Pisum sativum*) seedlings from 5 to 10 percent. The significance of the increased chlorophyll is not known. Some species of lichens exposed to 3,760 $\mu g/m^3$ (2.0 ppm) for 6 hours showed reduced chlorophyll content.

In contrast to studies cited on the effects of NO_2 alone, a number of studies on mixtures of NO_2 and SO_2 showed that the NO_2 injury threshold was significantly decreased and that the effects of the two gases in combination were at least additive and usually more than additive. Concentrations at which observable injury occurred were well within the ambient concentrations of NO_2 and SO_2 occurring in some areas of the U.S. A combination of 188 $\mu g/m^3$ (0.1 ppm) NO_2 and 262 $\mu g/m^3$ (0.1 ppm) SO_2 for 4 hours caused moderate leaf injury in pinto bean (*Phaseolus vulgaris*), radish (*Raphanus sativus*), soybean (*Glycine max*), tomato (*Lycopersicon esculentum*), oat (*Avena sativa*), and tobacco (*Nicotiana tabacum*). Exposure to 282 $\mu g/m^3$ (0.15 ppm) NO_2 in combination with 262 $\mu g/m^3$ (0.1 ppm) SO_2 for 4 hours caused more injury. Neither 3,760 $\mu g/m^3$ (2.0 ppm) NO_2 nor 1,310 $\mu g/m^3$ (0.5 ppm) SO_2 alone caused injury. Research data from grass species exposed for 20 weeks to concentrations of 210 $\mu g/m^3$ (0.11 ppm) NO_2 and 290 $\mu g/m^3$ (0.11 ppm) SO_2 for 103.5 hours per week showed significant reductions in yield parameters ranging from 30 to 90 percent indicating that concentrations of these two gases occurring simultaneously can have major deletrious effects on plant growth.

1.4.3 Effects of Nitrogen Oxides on Materials

The damaging effects of atmospheric oxides of nitrogen have been established for a variety of materials including dyes, fibers, plastics, rubber, and metals as discussed in Chapter 13 of this document.

Field exposures of cotton, viscose rayon, cellulose acetate, and nylon fabrics colored with representative dyes demonstrate that fading occurs for specific dyes in air containing NO_2, O_3, and SO_2. These exposures were carried out in ambient air and protected against sunlight. Chamber studies using individual pollutants NO_2, O_3, and SO_2 have shown that some individual dye-fiber combinations exhibit color fading only in response to NO_2 exposure, whereas others are susceptible to O_3, as well as combinations of NO_2 and O_3. SO_2 introduced an accelerant effect. Disperse dyes used for cellulose acetate and rayon include vulnerable anthraquinone blues and reds. The cellulosic fibers cotton and viscose rayon, dyed with certain widely used direct dyes, vat dyes, and fiber reactive dyes, suffer severe fading on chamber exposures to 940 $\mu g/m^3$ (0.5 ppm) NO_2 under high humidity (90 percent) and high temperature (90°F) conditions. Significant fading is observed on 12 weeks exposure to 94 $\mu g/m^3$ (0.05 ppm) NO_2 under high humidity and temperature conditions (90 percent, 90°F).

Acid dyes on nylon fade on exposure to NO_2 at levels as low as 188 $\mu g/m^3$ (0.1 ppm), under similar conditions. Dyed polyester fabrics are highly resistant to NO_2-induced fading. However, permanent press fabrics of polyester cotton and textured polyester exhibited unexpected fading when first marketed. The fading was in the disperse dye which migrated under high heat conditions of curing or heat setting to the reactive medium of resins and other surface additives.

The yellowing of white fabrics is documented for polyurethane segmented fibers (Lycra and Spandex), rubberized cotton, optically brightened acetate, and nylon. Yellowing is also reported on fabrics which were finished with softeners or anti-static agents. Nitrogen dioxide was demonstrated to be the pollutant responsible for color change, with O_3 and SO_2 showing no effect. Chamber studies using NO_2 concentrations of 376 $\mu g/m^3$ (0.2 ppm) for 8 hours showed yellowing equivalent to that on garments returned to manufacturers.

The tensile strength of fabrics may be adversely affected by the hydrolytic action of acid aerosols. Nitrogen dioxide has been demonstrated to oxidize the terminal amine group ($-NH_2$) of nylon to the degree that the fiber has less affinity for acid-type dyes. Nylon 66 may suffer chain scission when exposed to 1,880 to 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm) NO_2. Field exposures of fibers emphasize the action of acids derived from SO_2, although NO_2 may also be present in high concentrations in urban sites. Information on the contribution of NO_2 to degradation is incomplete.

Although a survey of the market for plastics predicts the use of 1.78 billion pounds in 1982, there is very little information on the effects of NO_2 on polyethylene, polypropylene, polystyrene, polyvinylchloride, polyacrylonitrile, polyamides and polyurethanes. Aging tests involve sunlight exposure as well as exposure to ambient air. Chamber exposure of the above plastics to combinations of SO_2, NO_2, and O_3 has resulted in deterioration. Nitrogen dioxide alone has caused chain scission in nylon and polyurethane at concentrations of 1,880 and 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm).

The extensive data on corrosion of metals in polluted areas relate the corrosion effects to the SO_2 concentrations. The presence of NO_2 and its contribution is not evaluated despite its presence as acid aerosol in appreciable concentrations.

Ammonium nitrates were implicated as a factor in the stress corrosion cracking of wires made of nickel brass alloy used in telephone equipment. Since nitrate salts have been shown to be more hygroscopic than either chloride or sulfate salts, the presence of nitrates may lower the humidity requirements for the formation of an aqueous electrolyte system in the wet corrosion of metals.

1.4.4 Effects of Nitrogen Oxides on Visibility

As discussed in Chapter 10, regarding NO_x effects on visibility, ambient air pollution degrades the appearance of distant objects and reduces the range at which they can be distinguished from the background. These effects are manifest not only in visible plumes, but also in large-scale, hazy air masses. Haze and plumes can result in the discoloration, deterioration, and loss of scenic vistas, particularly in areas of the southwestern United States where visibility is generally good. Under extreme conditions reduced visual range and contrast due to haze and plumes may impede air traffic. NO_2 does not significantly reduce visual range; however, NO_2 can be responsible for a portion of the brownish coloration observed in polluted air. It should be noted that non-nitrate particulate matter has also been implicated in the production of a significant portion of brownish coloration. Under certain circumstances, brown plumes may be distinguished tens of kilometers downwind of their sources.

Nitrogen dioxide in the atmosphere acts as a blue-minus filter for transmitted light. It tends to impart a brownish color to targets, including the sky viewed through the plume. The strength of this filter effect is determined by the amount of NO_2 concentration along the sight path; i.e., theoretically, similar effects are exerted by a 1 kilometer-wide plume containing 0.1 ppm (190 $\mu g/m^3$) of NO_2 or a 0.1 kilometer-wide plume containing 1.0 ppm (1,900 $\mu g/m^3$) of NO_2. Less than 0.1 ppm-km NO_2 is sufficient to produce a color shift which is distinguishable in carefully controlled, color-matching tests. Reports from one laboratory using NO_2-containing sighting tubes indicate a visible color threshold of 0.06 ppm-km for the typical observer. This value was supported by a few field observations of NO_2 plumes from nitric acid manufacturing plants under varying operating conditions. The value cited refers to the effect of NO_2 in the absence of atmospheric aerosol. Empirical observations under a variety of conditions are needed to determine the perceptibility of NO_2 in ambient air.

Plume coloration due to NO_2 is modified by particulate matter and depends on a number of factors such as sun angle, surrounding scenery, sky cover, viewing angle, human perception parameters, and pollutant loading. Suspended particles generally scatter in the forward direction and can thus cause a haze layer or a plume to appear bright in forward scatter (sun in front of the observer) and dark in back scatter (sun in back of the observer) because of the angular variation in scattered airlight. This effect can vary with background, sky, and objects. Aerosol optical effects alone are capable of imparting a reddish brown color to a haze layer when viewed in backward scatter. NO_2 would increase the degree of coloration in such a situation. When the sun is in front of the observer, however, light scattered toward him by the plume tends to washout the brownish light transmitted from beyond. Under these conditions, light scattering by particles diminishes the plume coloration caused by NO_2. Estimates of the magnitude of this effect attributable to particulate nitrates are currently hampered by the lack of data on particulate nitrate concentrations in ambient air.

2. INTRODUCTION

Molecular nitrogen (N_2) and oxygen (O_2) are normal constituents of the air we breathe. Together they comprise well over 90 percent of the earth's atmosphere, and both are essential to life. As a gas in the atmosphere, O_2 is vital to the respiration of all life forms except the anaerobes. N_2 is essentially inert in all but the nitrogen-fixing organisms. Through the action of natural or man-made processes, however, the two elements can combine with each other or with other elements to form toxic compounds. This air quality criteria document compiles in a single source document available information about the formation and occurrence of such compounds in the atmosphere, and evidence of their effects on man and the biosphere.

The initial Air Quality Criteria for Nitrogen Oxides (EPA Publication No. AP-84) was published by the U.S. Environmental Protection Agency (USEPA) in 1971. The information presented provided the basis for the present annual air quality standard for nitrogen dioxide (NO_2), an arithmetic average not to exceed 100 $\mu g/m^3$ (0.05 ppm). At the time, insufficient information was available to support promulgation of a short-term standard. However, since the annual standard does not preclude short-term peak NO_2 concentrations which may be harmful, the Congress, in the 1977 amendments to the Clean Air Act, required that the air quality criteria document for nitrogen oxides (NO_x) be revised. In addition to the question of short-term exposure, this document is required to address nitric and nitrous acids, nitrates, nitrites, nitrosamines and other derivatives of oxides of nitrogen. NO_2 is the principal subject of this document, since available evidence indicates it is the nitrogenous compound of most concern for human health and welfare. However, the health and welfare effects of other airborne nitrogen compounds including other nitrogen oxides, nitrogen acids, nitrates, nitrites, nitrosamines and other derivative compounds are also presented. Also included are descriptions of the complex chemical reactions occurring in polluted atmospheres that link, to some extent, the atmospheric concentrations of nitrogen oxides with the presence of photochemical oxidants such as ozone. The ozone/NO_x relationship is discussed in greater detail in the Air Quality Criteria for Ozone and Other Photochemical Oxidants, EPA-600/8-78-004.

Nitrogen oxides are produced when fossil fuels are burned, both by the oxidation of nitrogen in the fuel and by the high-temperature oxidation of atmospheric nitrogen. Most NO_x emissions occur as nitric oxide (NO). Available evidence indicates that nitric oxide in the ambient air is not of direct concern for human health and welfare. NO is, however, further oxidized in the amosphere to a variety of other nitrogenous compounds. Of these, NO_2 is the compound of most concern.

Increases in the U.S. consumption of energy will stimulate increasing combustion of fossil fuels which can result in an increase in NO_x emissions. Atmospheric concentrations of NO_2 and other nitrogenous compounds are, therefore, likely to become higher in the future, especially if NO_x emissions control actions do not keep pace with energy production.

Considerable information has been developed since publication of the original Air Quality Criteria for Nitrogen Oxides in 1971. Information reviewed in this document which demonstrates the toxicity of a number of nitrogen compounds has been derived from a variety of human, animal, and ecological studies. For example, studies of both animals and humans demonstrate relationships between exposure to NO_2 and various aspects of pulmonary function, particularly those types of physiological or pathological changes that may lead to the development of chronic respiratory disease. Other studies have been concerned with the effect of NO_2 exposure on susceptibility to acute respiratory disease. Adverse effects of nitrogen compounds on plants and inanimate materials have also been reported.

The determination of the effects of exposure to airborne nitrogen compounds on human health encounters four major difficulties. The first is that nitrogen compounds comprise only a portion of a complex of pollutants in the ambient air. Adverse effects found in epidemiological studies may result from exposure to individual compounds or a combination of multiple compounds. Epidemiologists, evaluating community studies, have not been able to assess unequivocally the effects of exposure to individual compounds. Consequently, it is useful to use the combined results of epidemiological and animal studies to assess potential harmful effects. Animal studies reported in this document show the effects of exposure to individual compounds as well as certain effects of the compounds in combination. In some cases, these studies also show, for individual animal species, the maximum dose of the pollutant tolerated, target organs, mechanisms of action, and lowest effective dose. In addition, the studies can show whether there is a consistency of effects across a variety of animal species.

The second problem is the difficulty of assessing, with a high degree of accuracy, the actual day-to-day exposure to NO_x of individuals or populations in ambient situations. Because there are a limited number of monitoring stations, air measurement data are not completely representative of actual human exposure. This difficulty is compounded by the mobility of the population, the portion of each day spent inside buildings, and the variability of atmospheric concentrations of pollutants over short distances.

The third problem encountered in determining the effects of exposure to nitrogen compounds is that dose/response data derived from animal studies cannot readily be extrapolated to humans. Indications of probable effects on humans can be obtained from the animal studies, and a consistent effect among animal species, especially when primates are included, increases confidence that a similar effect may occur in humans.

The fourth problem is the determination of effective exposure time. NO_x community studies usually use data on annual or daily mean levels of exposure. Only occasionally are hourly values provided. Based on animal studies, however, it may be inferred that repeated intermittent exposure to daily peak values may be more significant in the production of adverse health effects than is an equivalent or even greater total dose delivered by continuous exposure to the observed long-term averages. If this is true, the protection of human health is achieved more effectively by reducing the peak concentrations rather than the long-term means. The subject of effective control strategies, however, is outside the scope of this document.

In response to the 1977 Clean Air Act Amendments, this document provides a summarization of available data relevant to the effects on human health and welfare of exposure to nitrogen oxides or other toxic materials evolving from nitrogen oxides in the atmosphere. Various sections of the document provide information on (1) the intensity and frequency, in this country, of the occurrence of significant atmospheric concentrations of toxic nitrogenous compounds and their sources, (2) the results of animal studies concerned with the effect of short- or long-term exposures to these compounds, (3) the results of controlled human exposure studies, (4) the results of community exposure studies, (5) the results of studies of the effects of atmospheric nitrogenous compounds on visibility, ecologic systems, plants, and materials and (6) the relationship of these compounds to large-scale phenomena such as acidic precipitation and perturbations of the stratospheric ozone layer.

This document does not constitute a detailed literature review of the subjects covered. Not every published manuscript is cited; however, major publications relevant to the topics covered are included.

In reviewing and summarizing the literature, an attempt has been made to present alternative points of view where scientific controversy exists. In some instances, considerations bearing on the quality of studies have been included. The needs for subsequent studies have not, for the most part, been addressed.

Chapter 1 summarizes those effects on human health and welfare which are considered of most concern, and through interpretation of study results, defines, to the degree possible, the pollutant concentration levels at which adverse effects are discernible. Other chapter summaries appear at the ends of individual chapters covering information not presented in Chapter 1.

As is appropriate in a criteria document, the discussion is descriptive of the range of exposures and the attendant effects. Information is presented, and the evidence is evaluated, but no judgments are made concerning the maximum levels of exposure that should be permitted. Such judgments would be recommendations concerning air quality standards and management, which are prescriptive in nature, and not within the purview of this document.

3. GENERAL CHEMICAL AND PHYSICAL PROPERTTIES OF NO_x AND NO_x-DERIVED POLLUTANTS

3.1 INTRODUCTION AND OVERVIEW

In this chapter some general chemical and physical properties of NO_x* and NO_x-derived pollutants are discussed by way of introduction to the complex chemical and physical interactions which may occur in the atmosphere and other media. The discussion will be significantly augmented throughout the document as particular topics are discussed in depth.

There are eight oxides of nitrogen (NO_x) that may be present in the ambient air: nitric oxide (NO), nitrogen dioxide (NO_2), nitrous oxide (N_2O), unsymmetrical nitrogen trioxide (OONO), symmetrical nitrogen trioxide (ON(O)O), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and dinitrogen pentoxide (N_2O_5).

Of these, NO and NO_2 are generally considered the most important in the lower troposphere because they may be present in significant concentrations (Chapter 8). Their interconvertibility in photochemical smog reactions (Chapter 6) has frequently resulted in their being grouped together under the designation NO_x, although analytic techniques can distinguish clearly between them (Chapter 7). Of the two, NO_2 is the more toxic and irritating compound (Chapter 14 and 15).

Nitrous oxide is ubiquitous even in the absence of anthropogenic sources, since it is a product of natural biologic processes in soil (Chapters 4 and 12). It is not known, however, to be involved in any photochemical smog reactions. Although N_2O is not generally considered to be an air pollutant, it participates in upper atmospheric reactions involving the ozone layer (Chapter 9).

While OONO, ON(O)O, N_2O_3, N_2O_4, and N_2O_5 may play a role in atmospheric chemical reactions leading to the transformation, transport, and ultimate removal of nitrogen compounds from ambient air (Chapter 6), they are present only in very low concentrations, even in polluted environments.

Ammonia (NH_3) is generated, on a global scale, during the decomposition of nitrogenous matter in natural ecosystems and it may also be produced locally in larger concentrations by human activities such as the maintenance of dense animal populations (Chapter 4). It is discussed briefly in this document to facilitate understanding of the nitrogen cycle and also because some researchers have suggested that NH_3 is converted to NO_x in the atmosphere.

Other NO_x-derived compounds which may be found in polluted air include nitrites, nitrates, nitrogen acids, N-nitroso compounds, and organic compounds such as the peroxyacyl nitrates

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*For all practical purposes, NO_x is the sum of nitrogen dioxide (NO_2) and nitric oxide (NO).

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($RC(O)OONO_2$, where R represents any one of a large variety of possible organic groups) (Chapter 8).

The peroxyacyl nitrates, of which peroxyacetyl nitrate ($CH_3C(O)OONO_2$, or PAN) is of most concern in terms of atmospheric concentrations, have been thoroughly reviewed in the recent EPA document, Air Quality Criteria for Ozone and Other Photochemical Oxidants (1978) and will be given only the briefest discussion in this chapter and elsewhere in this document.

Recent discovery of N-nitroso compounds in air, water, and food has led to concern about possible human exposure to this family of compounds, some of which have been shown to be carcinogenic in animals. Health concerns also have been expressed about nitrates, which occur as a component of particulate matter in the respirable size range, suspended in ambient air (Chapter 15). Some of this particulate nitrate is produced in atmospheric reactions (Chapter 6). Nitrates may also occur in significant concentrations in drinking water supplies but this occurrence is not believed to be the result of atmospheric production.

Photochemical models predict that up to one-half of the original nitrogen oxides emitted may be converted on a daily basis to nitrates and nitric acid (HNO_3). This atmospheric production of nitric acid is an important component of acid rain (Chapter 11).

Table 3-1 summarizes current theoretical estimates of the concentrations of the various nitrogen oxides and acids that would be present in an equilibrium state assuming initially only molecules of nitrogen and oxygen at 1 atm pressure, 25°C and 50 percent relative humidity. The low concentrations of many of the oxides and acids preclude direct measurement of most of them in the ambient air. Consequently, most studies leading to predictions of concentrations rely on theoretical estimates derived from small-scale laboratory studies.

In fact, the thermodynamic equilibrium state is not achieved in polluted, sunlight-irradiated atmospheres. Rather, expected concentrations of pollutants are influenced by emissions and subsequent reactions and tend to be much greater than those at equilibrium. Table 3-1 lists one set of estimated concentrations of nitrogen oxides and acids expected under more realistic conditions, derived from computer simulations of photochemical smog reactions which might occur in more or less typical urban environments.

3.2 NITROGEN OXIDES

Table 3-2 summarizes some important physical properties of nitrogen oxides under standard temperature and pressure (STP) conditions of 25°C and 1 atm, respectively. The remainder of this section describes chemical and physical properties of individual nitrogen oxide species.

3.2.1 Nitric Oxide (NO)

Nitric oxide is an odorless gas. It is also colorless since its absorption bands are all at wavelengths less than 230 nm, well below the visible wavelengths (Figure 3-1). Nitric oxide is only slightly soluble in water (0.006 g/100 g of water at 24°C and 1 atm pressure).

TABLE 3-1. THEORETICAL CONCENTRATIONS OF NITROGEN OXIDES AND NITROGEN ACIDS WHICH WOULD BE PRESENT AT EQUILIBRIUM WITH MOLECULAR NITROGEN, MOLECULAR OXYGEN, AND WATER IN AIR AT 25°C, 1 ATM, 50 PERCENT RELATIVE HUMIDITY (Demerjian et al., 1974)

Compound	Concentrations in Hypothetical Atmosphere, ppm	
	At Equilibrium	In Typical Sunlight-irradiated, Smoggy Atmosphere[a]
O_2	2.06×10^{5}	2.06×10^{5}
N_2	7.69×10^{5}	7.69×10^{5}
H_2O	1.56×10^{4}	1.56×10^{4}
NO_2	1.91×10^{-4}	10^{-1}
NO	2.69×10^{-10}	10^{-1}
NO_3	3.88×10^{-16}	10^{-8}-10^{-9}
N_2O_3	2.96×10^{-20}	10^{-8}-10^{-9}
N_2O_4	2.48×10^{-13}	10^{-7}-10^{-8}
N_2O_5	3.16×10^{-17}	10^{-3}-10^{-5}
HONO (cis)	7.02×10^{-9}	10^{-3}
HONO (trans)	1.60×10^{-8}	10^{-3}
$HONO_2$	1.33×10^{-3}	10^{-2}-10^{-3}

[a] Theoretical estimates made using computer simulations of the chemical reactions rates in a synthetic smog mixture.

TABLE 3-2. SOME PHYSICAL AND THERMODYNAMIC PROPERTIES OF THE NITROGEN OXIDES

Oxide	Molecular Weight, g/mol	Melting Point $^{o}C^{a,b}$	Boiling Point $^{o}C^{a,b}$	Solubility in $H_2O(0^{o}C)$, cm^3 (STP)/100 g^a	Thermodynamic Functions (Ideal Gas, 1 atm, 25^{o}C)^c Enthalpy of Formation, kcal/mol	Thermodynamic Functions (Ideal Gas, 1 atm, 25^{o}C)^c Entropy, cal/mol-deg
NO	30.01	-163.6	-151.7	7.34	21.58	50.347
NO_2	46.01	Liquid, solid forms largely as N_2O_4		Reacts with H_2O forming $HONO_2$ and HONO	7.91	57.34
N_2O_4	92.02	-11.3	21.2	Reacts with H_2O forming $HONO_2$ and HONO	2.17	72.72
N_2O	44.02	-102.4	-89.5	130.52	19.61	52.55
N_2O_3	76.02	-102	3.5 (decomposes)	Reacts with H_2O forming $HONO_2$	19.80	73.91
N_2O_5	108.01	30	32.4 (decomposes)	Reacts with H_2O forming $HONO_2$	2.7	82.8

[a] Matheson Gas Data Book (Matheson, 1966).
[b] Handbook of Chemistry and Physics (Chemical Rubber Company, 1969-1970).
[c] JANAF Thermochemical Tables (U. S. Department of Commerce, 1971).

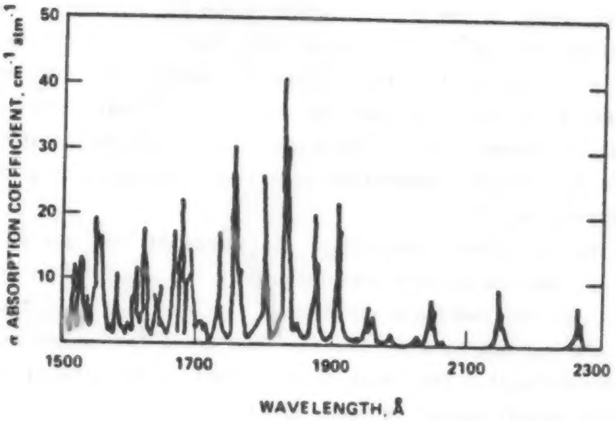

Figure 3-1. Absorption spectrum of nitric oxide (McNesby and Okabe, 1964).

It has an uneven number of valence electrons, but, unlike NO_2, it does not dimerize in the gas phase.

Nitric oxide is a principal byproduct of combustion processes, arising from the oxidation of molecular nitrogen in the combustion air and of organically bound nitrogen present in certain fuels such as coal and heavy oil. The oxidation of nitrogen in combustion air occurs primarily through a set of reactions known as the extended Zeldovitch mechanism (Zeldoritch, 1946):

$$N_2 + O \rightarrow NO + N$$

$$N + O_2 \rightarrow NO + O$$

with the additional equation (extended mechanism)

$$N + OH \rightarrow NO + H$$

The high activation energy of the first reaction above (75 kcal/mol) coupled with its essential function of breaking the strong N_2 triple bond make this the rate limiting step of the Zeldovitch mechanism. Due to the high activation energy, this mechanism for NO production proceeds at a somewhat slower rate than the reactions of fuel constituents and is extremely temperature sensitive (Bowman, 1973). Moreover, the production of atomic oxygen required for the first step is also highly temperature sensitive. NO formed via this mechanism is often referred to as "thermal NO_x."

In addition to the strong temperature dependence of the rate of the first step of the Zeldovich mechanism, the temperature also influences the amount of atomic oxygen (O) available for the reaction. In the immediate vicinity of a flame, the high temperatures coupled with the kinetics of the hydrocarbons in the fuel can drive the oxygen concentration to several times its equilibrium level. The local ratio of fuel to air also has a first order effect on the concentration of atomic oxygen (Bowman, 1973).

The reaction kinetics of thermal NO formation is further complicated by the fact that certain hydrocarbon radicals can be effective in splitting the N_2 bond through reactions such as (Fenimore, 1976):

$$CH + N_2 \rightarrow CHN + N$$

The rate of oxidation of the fuel (and intermediate hydrocarbon radical fragments) is usually sufficiently rapid that only negligible quantities of the fuel radicals are available to attack the molecular nitrogen. However, under fuel-rich conditions, this can become the dominant mode of breaking the N_2 bond and, in turn, can be responsible for significant NO formation (Engleman et al., 1976). Such reactions appear to have a relatively low activation energy and can proceed at a rate comparable to oxidation of the fuel. Because of the early formation of NO by this mechanism, relative to that formed by the Zeldovitch mechanism, NO thus formed is often referred to as "prompt NO." The importance of this mechanism has not been quantified for practical systems.

In fuels such as coal and residual fuel oil, nitrogen compounds are bound within the fuel matrix. Typically, Number 6 residual oil contains 0.2 to 0.8 percent by weight bound nitrogen and coal typically contains 1 to 2 percent. If this 1 percent nitrogen were converted quantitatively to NO_x, it would account for about 2,000 ppm NO_x in the exhaust of a coal-fired unit. In practice, only a portion of these nitrogen compounds is converted to NO_x, with the remainder being converted to molecular nitrogen (N_2). Tests designed to determine the percent of the NO_x emissions due to oxidation of bound nitrogen (Pershing and Wendt, 1976) show that upward of 80 percent of the NO_x from a coal-fired boiler originate from this source. Details of the kinetic mechanisms involved in fuel nitrogen oxidation are uncertain due in part to the variability of molecular composition among the many types (and sources) of coal and heavy oils and to the complex nature of the heterogeneous processes occurring. Experimental evidence does, however, lend some insight into the processes involved. A number of fuel-bound nitrogen compounds have been cited (Axworthy and Schuman, 1973; Martin et al., 1971; Turner and Siegmund, 1972), but the degree of conversion to NO_x does not seem to be significantly affected by the compound type. NO_x conversions arising from fuel sources seem also to be relatively insensitive to temperature in diffusion flames. The most important parameters in determining fuel-bound nitrogen conversion appear to be the local conditions prevailing when the nitrogen is evolved from the fuel. Under fuel-rich conditions this nitrogen tends to form N_2, whereas under fuel-lean conditions significant amounts of NO_x are formed.

Nitric oxide formation kinetics in typical furnaces are not fast enough to reach equilibrium levels in the high temperature flame zone, while the NO destruction mechanisms are far too slow to allow the NO, once formed, to reach equilibrium at typical stack temperatures. This is to say that the NO formation process is kinetically controlled.

Nitric oxide and nitrogen dioxide produced in relatively large concentrations at high temperatures in combustion processes would revert to lower concentrations characteristic approximately of the equilibrium values shown in Table 3-3 were it not for the fact that combustion equipment rapidly converts a large fraction of the thermal energy available to useful work. This results in a rapid cooling of the combustion gases and a "freezing-in" of the produced NO and NO_2 near concentrations characteristic of the high temperature phase of the process.

A major implication of the fact that NO_x emissions are defined by the kinetics of the process rather than being an equilibrium phenomenon is that NO_x emissions can be effectively modified by changes in the details of the combustion process. For clean fuels such as natural gas or Number 2 distillate oil with no bound nitrogen, the NO formation is dominated by the Zeldovitch mechanism. Thus, combustion modifications which reduce peak flame temperature, limit the gas residence time at peak temperatures and/or reduce the amount of atomic oxygen available at high temperatures will reduce the NO_x emissions. Examples of such modifications

TABLE 3-3. THEORETICAL EQUILIBRIUM CONCENTRATIONS OF NITRIC OXIDE AND NITROGEN DIOXIDE IN AIR (50 PERCENT RELATIVE HUMIDITY) AT VARIOUS TEMPERATURES (NATIONAL RESEARCH COUNCIL, 1977)

Temperature, ^{o}K (^{o}C)	Concentration, µg/m^3 (ppm)	
	NO	NO_2
298 (24.85)	3.29×10^{-10} (2.63×10^{-10})	3.53×10^{-4} (1.88×10^{-4})
500 (226.85)	8.18×10^{-4} (6.54×10^{-4})	7.26×10^{-2} (3.86×10^{-2})
1,000 (726.85)	43 (34.4)	3.38 (1.80)
1,500 (1,226.85)	1,620 (1,296)	12.35 (6.57)
2,000 (1,726.85)	9,946.25 (7,957)	23.88 (12.70)

are flue gas recirculation, reduced load, reduced combustion air preheat temperature, water injection and reduced excess air (Proceedings, 1976; Proceedings, 1977).

In furnaces fired with coal or heavy oil, the major portion of the NO_x emissions is from fuel-bound nitrogen conversion. Thus, combustion modifications which reduce the availability of oxygen when the nitrogen compounds are evolved will reduce the NO_x produced. Examples of such modifications are reduction of the amount of excess air during firing, establishing fuel-rich conditions during the early stages of combustion (staged combustion), or new burner designs that tailor the rate of mixing between the fuel and air streams (Proceedings, 1976).

3.2.2 Nitrogen Dioxide (NO_2)

Nitrogen dioxide is a reddish-orange-brown gas with a characteristic pungent odor. Although its boiling point is 21.1°C, the low partial pressure of NO_2 in the atmosphere prevents condensation. Nitrogen dioxide is corrosive and highly oxidizing. It has an uneven number of valence electrons and forms the dimer N_2O_4 at higher concentrations and lower temperatures, but the dimer is not important at ambient concentrations. In the atmosphere NO can be oxidized to NO_2 by the thermal reaction:

$$2\ NO + O_2 \rightarrow 2\ NO_2$$

However, this reaction is of minor importance in most urban ambient situations, since other chemical processes are faster. The above reaction is mainly responsible for the NO_2 present in combustion exhaust gases. About 5 to 10 percent by volume of the total emissions of NO_x from combustion sources is in the form of NO_2, although substantial variations from one source to another have been observed. Under more dilute ambient conditions, photochemical smog reactions involving hydrocarbons convert NO to NO_2 (Chapter 6).

Nitrogen dioxide's principal involvement in photochemical smog stems from its absorption of sunlight and subsequent decomposition (photolysis) to NO and atomic oxygen (O). Nitrogen dioxide is an efficient absorber of light over a broad range of ultraviolet and visible wavelengths. Only quanta with wavelengths less than about 430 nm, however, have sufficient energy to cause photolysis. It should also be noted that photons having wavelengths less than about 290 nm are largely absorbed in the upper atmosphere. The effective range of wavelengths responsible for photolysis of NO_2 at ground level is, therefore, 290 nm to 430 nm. Because of its absorption properties, NO_2 produces discoloration and reduces visibility in the polluted lower troposphere.

3.2.3 Nitrous Oxide (N_2O)

Nitrous oxide is a colorless gas with a slight odor at high concentrations. Nitrous oxide in the atmosphere arises as one product of the reduction of nitrate by a ubiquitous group of bacteria that use nitrate as their terminal electron acceptor in the absence of oxygen (denitrification) (Brezonik, 1972; Delwiche, 1970; Focht and Verstraete; Kenney, 1973).

Although N_2O does not play a significant role in atmospheric reactions in the lower troposphere, it participates in a mechanism for ozone decomposition in the stratosphere.

Nitrous oxide transported to the stratosphere undergoes photolysis by absorbing ultraviolet (UV) radiation at wavelengths below 300 nm to produce N_2 and singlet oxygen (Johnston and Selwyn, 1975):

$$N_2O + h\nu \rightarrow N_2 + O(^1D)$$ where hv is a unit of radiant energy

Singlet oxygen, which also is produced by ozone photolysis, reacts with more nitrous oxide to produce two sets of products:

$$N_2O + O(^1D) \rightarrow N_2 + O_2$$

and

$$\rightarrow NO + NO$$

The NO produced enters a catalytic cycle, the net result of which is the regeneration of NO_x and the destruction of ozone:

$$NO + O_3 \rightarrow NO_2 + O_2$$
$$O_3 + h\nu \rightarrow O_2 + O$$
$$NO_2 + O \rightarrow NO + O_2$$

These reactions are of concern because of the possibility that increased N_2O resulting from denitrification of excess fertilizer may lead to a decrease of stratospheric ozone (Cast, 1976; Crutzen, 1976) with consequent potential for adverse human health effects.

3.2.4 Unsymmetrical Nitrogen Trioxide (OONO)

Unsymmetrical nitrogen trioxide is thought to be an intermediate in the reaction of NO with O_2:

$$NO + O_2 \rightleftarrows O\text{-}O\text{-}N\text{-}O$$
$$O\text{-}O\text{-}N\text{-}O + NO \rightarrow 2NO_2$$

There is, however, no direct evidence for the existence of this species. If it does exist, it is, nevertheless, of little importance in the chemistry of polluted atmospheres, since the NO/O_2 reaction accounts for very little of the NO oxidized.

3.2.5 Symmetrical Nitrogen Trioxide (NO_3)

Symmetrical nitrogen trioxide has been identified in laboratory systems containing NO_2/O_3, NO_2/O, and N_2O_5 as an important reactive transient (Johnston, 1966). It is likely to be present in photochemical smog. This compound can be formed as follows:

$$O_3 + NO_2 \rightarrow NO_3 + O_2$$
$$O + NO_2\ (+ M) \rightarrow NO_3\ (+ M)$$
$$N_2O_5\ (+ M) \rightarrow NO_3 + NO_2\ (+ M)$$

(where M represents any third molecule available to remove a fraction of the energy involved in the reaction.)

Symmetrical nitrogen trioxide is highly reactive towards both nitric oxide and nitrogen dioxide.

$$NO_3 + NO \rightarrow 2NO_2$$
$$NO_3 + NO_2 \ (+ M) \rightarrow N_2O_5 \ (+ M)$$

Its expected concentration in polluted air is very low (about 10^{-6} µg/m^3 or 10^{-9} ppm).

3.2.6 Dinitrogen Trioxide (N_2O_3) (Also Known as Nitrogen Sesquioxide)

In the atmosphere, N_2O_3 is in equilibrium with NO and NO_2 according to the following equation:

$$NO + NO_2 \rightleftarrows N_2O_3$$

The equilibrium concentrations at typical urban levels of NO and NO_2 range from about 10^{-4} µg/m^3 (~10^{-7} ppm) to 10^{-6} µg/m^3 (~10^{-9} ppm) (Table 3-4). N_2O_3 is the anhydride of nitrous acid and reacts with liquid water to form the acid:

$$N_2O_3 + H_2O \rightarrow 2HONO$$

3.2.7 Dinitrogen Tetroxide (N_2O_4) (Also Known as Nitrogen Tetroxide)

Dinitrogen tetroxide is the dimer of NO_2 formed by the association of NO_2 molecules. It also readily dissociates to establish the equilibrium:

$$2NO_2 \rightleftarrows N_2O_4$$

Table 3-4 presents theoretical predictions of concentrations of N_2O_3 and N_2O_4 in equilibrium with various NO and NO_2 concentrations.

3.3 NITRATES, NITRITES, AND NITROGEN ACIDS

Nitric acid in the gaseous state is colorless and photochemically stable. The major pathway for atmospheric formation of nitric acid is given by:

$\cdot OH + NO_2 \rightarrow HNO_3$ where $\cdot OH$ is a hydroxyl free radical

It is a volatile acid, so that at ambient concentrations in the atmosphere, the vapor would not be expected to coalesce into aerosol and be retained unless the aerosol contains reactants such as ammonia (NH_3) to neutralize the acid, producing particulate nitrates (Chapter 6).

The nitrate ion (NO_3^-) is the most oxidized form of nitrogen. Since nitrate is chemically unreactive in dilute aqueous solution, nearly all of the transformations involving nitrate in natural waters result from biochemical pathways. The nitrate salts of all common metals are quite soluble.

Nitrates can be reduced to nitrites by microbial action. Many of the deleterious effects of nitrate result from its conversion to nitrite. The nitrite ion represents an intermediate and relatively unstable oxidation state (+3) for nitrogen. Both chemical and biological processes can result in its further reduction to various products, or its oxidation to nitrate. Nitrite salts are also quite soluble.

TABLE 3-4. THEORETICAL CONCENTRATIONS OF DINITROGEN TRIOXIDE AND DINITROGEN TETROXIDE IN EQUILIBRIUM WITH VARIOUS LEVELS OF GASEOUS NITRIC OXIDE AND NITROGEN DIOXIDE IN AIR AT 25°C (NATIONAL RESEARCH COUNCIL, 1977)

Concentration, ppm			
NO	NO_2	N_2O_3	N_2O_4
0.05	0.05	1.3×10^{-9}	1.7×10^{-8}
0.10	0.10	5.2×10^{-9}	6.8×10^{-8}
0.50	0.50	1.3×10^{-7}	1.7×10^{-6}
1.00	1.00	5.2×10^{-7}	6.8×10^{-6}

The nitrite ion is the Lewis base of the weak acid, nitrous acid (HNO_2). When NO and NO_2 are present in the atmosphere, HNO_2 will be formed as a result of the reaction:

$$NO + NO_2 + H_2O \rightarrow 2HNO_2$$

However, in sunlight-irradiated atmospheres, the dominant pathway for nitrous acid formation is:

$$\cdot OH + NO \rightarrow HONO$$

Atmospheric concentrations of HONO are limited by the reverse reaction:

$$HONO + h\nu \rightarrow \cdot OH + NO$$

Nitrous acid is a weak reducing agent and is oxidized to nitrate only by strong chemical oxidants and by nitrifying bacteria. Nitrous acid reacts with amino acids (the Van Slyke reactions) to yield N_2. The reaction of nitrous acid with secondary amines to form N-nitrosamines is discussed in Section 3.5.

3.4 AMMONIA (NH_3)

Ammonia is a colorless gas with a pungent odor. It is extremely soluble in water, forming the ammonium (NH_4^+) and hydroxy (OH^-) ions. In the atmosphere, ammonia has been reported (Soderlund and Svensson, 1976) to be converted into oxides of nitrogen when it reacts with hydroxyl free radicals ($\cdot OH$). Burns and Hardy (1975) report that ammonia is oxidized into nitrates and nitrites in the atmosphere, and in geothermal wells. In the stratosphere, ammonia can be dissociated by irradiation with sunlight at wavelengths below 230 nm (McConnell, 1973).

3.5 N-NITROSO COMPOUNDS

Organic nitroso compounds contain a nitroso group (-N=O) attached to a nitrogen or carbon atom. According to Magee (1971), N-nitroso compounds generally can be divided into two groups--one group includes the dialkyl, alkylaryl, and diaryl nitrosamines, and the other, alkyl and aryl nitrosamides.

The principal chemical reaction involved in the formation of N-nitrosamines is that of the secondary amines with nitrous acid. Nitrosation is effected by agents having the structure ONX, where X = O-alkoxyl, NO_2^-, NO_3^-, halogen, tetrafluoroborate, hydrogen sulfate or OH_2^+. The equilibrium reaction of nitrosonium ion (ON^+), nitrous acid and nitrite ion:

$$ON^+ + OH^- \rightleftarrows HNO_2 \rightleftarrows H^+ + NO_2^-$$

is shifted to the right at pH > 7. The simplest form of nitrosation of amines involves electrophilic attack by the nitrosonium ion and subsequent deprotonation.

Mirvish (1970) studied the kinetics of dimethylnitrosamine (DMN) nitrosation and pointed out that the chief nitrosating agent at pH 1 is dinitrogen trioxide, the anhydride of nitrous acid, which forms reversibly from two HNO_2 molecules. The formation of nitrosamines is dependent on the pK of the amine.

Nitroso compounds are characteristically photosensitive and the nitroso group is split by UV radiation. Gaseous nitrosamines may be denitrosated by visible light. The electron absorption spectra of several nitrosamines are given in the literature (Rao and Bhaskar, 1969); the characteristic spectra show a low intensity absorption maximum around 360 nm and an intense band around 235 nm. Nitrosamines show three relatively intense bands in the infrared region of 7.1-7.4, 7.6-8.6, and 9.15-9.55 μm. Nuclear magnetic resonance (NMR), infrared (IR), ultraviolet (UV), and mass spectrometry (MS) spectra have been reviewed by Magee et al. (1976).

Atmospheric reactions involving nitrosamines are discussed in Chapter 6.

3.6 SUMMARY

There are eight nitrogen oxides which may be present in the ambient air: nitric oxide (NO), nitrogen dioxide (NO_2), nitrous oxide (N_2O), unsymmetrical nitrogen trioxide (OONO), symmetrical nitrogen trioxide (O-N(O)-O), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and dinitrogen pentoxide (N_2O_5).

Of these, NO and NO_2 are generally considered the most important in the lower troposphere because they may be present in significant concentrations in polluted atmospheres. Their interconvertibility in photochemical smog reactions has frequently resulted in their being grouped together under the designation NO_x, although analytic techniques can distinguish clearly between them. Of the two, NO_2 is the more toxic and irritating compound.

Nitrous oxide is ubiquitous even in the absence of anthropogenic sources since it is a product of natural biologic processes in soil. It is not known, however, to be involved in any photochemical smog reactions. Although N_2O is not generally considered to be an air pollutant, it is a principal reactant in upper atmospheric reactions involving the ozone layer.

While OONO, ON(O)O, N_2O_3, N_2O_4, and N_2O_5 may play a role in atmospheric chemical reactions leading to the transformation, transport, and ultimate removal of nitrogen compounds from ambient air, they are present only in very low concentrations, even in polluted environments.

Ammonia (NH_3) originates on a global scale during the decomposition of nitrogenous matter in natural ecosystems but it may also be produced locally by human activities such as the maintenance of dense animal populations. Some researchers have suggested conversion of NH_3 to NO_x in the atmosphere.

Compounds derived from NO_x including nitrites, nitrates, nitrogen acids, N-nitroso compounds, and organic compounds such as the peroxyacyl nitrates [$RC(O)OONO_2$], where R represents any one of a large variety of possible organic groups, may also be found in polluted air.

The peroxyacyl nitrates, of which peroxyacetyl nitrate [$CH_3C(O)OONO_2$] or PAN is of most concern in terms of atmospheric concentrations, have been thoroughly reviewed in the recent EPA document, Air Quality Criteria for Ozone and Other Photochemical Oxidants.

Recent discovery of N-nitroso compounds (some of which have been shown to be carcinogenic in animals) in air, water, food and tobacco products, has led to concern about possible human exposure to this family of compounds. Health concerns also have been expressed about nitric acid vapor and other nitrates, occurring as a component of particulate matter in the respirable size range, suspended in ambient air. Some of these nitrates are produced in atmospheric reactions. Nitrates may also occur in significant concentrations in public and private drinking water, but this occurrence is not believed to be the result of atmospheric production.

Photochemical models predict that up to one-half of the original nitrogen oxides emitted may be converted on a daily basis to nitrates and nitric acid. This atmospheric production of nitric acid is an important component of acidic rain.

3.6.1 Nitrogen Oxides

Nitric oxide (NO) is an odorless and colorless gas. It is a major by-product of the combustion process, arising both from the oxidation of molecular nitrogen in the combustion air and of nitrogen compounds bound in the fuel molecule. The amount of NO formed from the oxidation of molecular nitrogen is dependent upon such parameters as peak flame temperature, quantity of combustion air, and gas residence time in the combustion chamber. The amount of NO arising from oxidation of fuel-bound nitrogen does not seem to depend significantly on either the type of nitrogen compound involved or the flame temperature, but instead upon the specific air-to-fuel ratio at various stages in combustion.

Nitrogen dioxide (NO_2) is produced in minor quantities in the combustion process (5 to 10 percent of the total oxides of nitrogen). In terms of significant atmospheric loading in populated areas, NO_2 arises mainly from the conversion of NO to NO_2 by a variety of chemical processes in the atmosphere. Nitrogen dioxide is corrosive and highly oxidizing. Its reddish-orange-brown color arises from its absorption of light over a broad range of visible wavelengths. Because of its strong absorption in this range (and also in the ultraviolet spectrum), NO_2 can cause visibility reduction and affect the spectral distribution of solar radiation in the polluted, lower atmosphere.

3.6.2 Nitrates, Nitrites, and Nitrogen Acids

Other compounds derived from oxides of nitrogen (NO_x) by means of atmospheric chemical processes include nitrites, nitrates, nitrogen acids, organic compounds such as the peroxyacyl nitrates, and, possibly, the N-nitroso compounds.

Nitric acid, a strong acid and powerful oxidizing agent, is colorless and photochemically stable in the gaseous state. Its high volatility prevents condensation into droplets in the atmosphere unless the droplets contain reactants such as ammonia which neutralize the acid. Atmospheric reactions such as this may result in the formation of particulate nitrates suspended in ambient air.

3.6.3 N-Nitroso Compounds

The N-nitroso family comprises a wide variety of compounds all containing a nitroso group (-N=O) attached to a nitrogen or carbon atom. Their formation in the atmosphere has been postulated to proceed through chemical reaction of amines with NO_x and NO_x-derivatives in gas phase reactions and/or through atmospheric reactions involving aerosols. Nitroso compounds are characteristically photosensitive and the nitroso group is split by the ultraviolet radiation in sunlight. Gaseous nitrosamines may also be denitrosated by visible light.

CHAPTER 3

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4. THE NITROGEN CYCLE

4.1 INTRODUCTION

This chapter presents a discussion of the nitrogen cycle, pointing out the principal pathways, sources and sinks of nitrogen compounds in the environment on a global scale. This discussion is intended to provide background and perspective for the discussions relating to nitrogen and nitrogen oxides as presented in the other chapters of this document. One of the concerns relating to the effects of nitrogen oxide emissions into the atmosphere is the extent to which these emissions impinge on the natural cycling of this important nutrient element.

In addition to the nitrogen cycle, this chapter discusses estimates of the flow of nitrogen through the various biogeochemical compartments or pools in the global cycling of nitrogen.

The impact of man's intervention into the nitrogen cycle is discussed in Chapters 5 and 6 which describe the sources and the environmental transport of nitrogen oxides. Atmospheric concentrations are discussed in Chapter 8, the possible effects on the ozone layer in Chapter 9, Chapter 11 is concerned with acidic precipitation, and perturbations of the nitrogen cycle as evidenced in escosystem effects are specifically discussed in Chapter 12.

4.2 THE NITROGEN CYCLE

The major source of nitrogen is the earth's atmosphere where, in molecular form, it is a major constituent (79 percent). The flow of nitrogen from the atmosphere and its transformations in the biosphere are regulated almost entirely by terrestrial and aquatic microorganisms (Alexander, 1977; Bolin and Arrhenius, 1977; Delwiche, 1970, 1977).

In general outline, the nitrogen cycle is identical in terrestrial, fresh water, and oceanic habitats; only the microorganisms which mediate the various transformations are different (Alexander, 1977; Chen et al., 1972; Kenney, 1973) (Figure 4-1). A discussion of the step-by-step cycling of nitrogen follows.

1. Biological Nitrogen Fixation - Atmospheric nitrogen gas (N_2) is transformed into ammonia (NH_3 or NH_4^+) or nitrates (NO_3^-) in which form it enters the food chain. The transformation is carried out by a wide variety of microorganisms. The microorganisms may be either symbiotic (living in the roots of leguminous plants) or nonsymbiotic (living independently in the soil) and the process may be accomplished under aerobic or anaerobic conditions.

2. Organic Nitrogen Formation (assimilation) -- Fixed nitrogen as either nitrates or ammonia is assimilated by plants and converted into organic molecules such as amino acids, proteins, nucleic acids and vitamins. Plants are eaten by animals and plant proteins are converted to animal proteins. In addition, carnivores consume other animals as a protein source. Nitrogen is bound in plant or animal protein until the organisms die, or as in the case of animals, certain products are excreted.

3. Deamination or Ammonification -- This is a two-step process, also termed mineralization, in which the excretion products of animals and the proteins in dead plants and animals

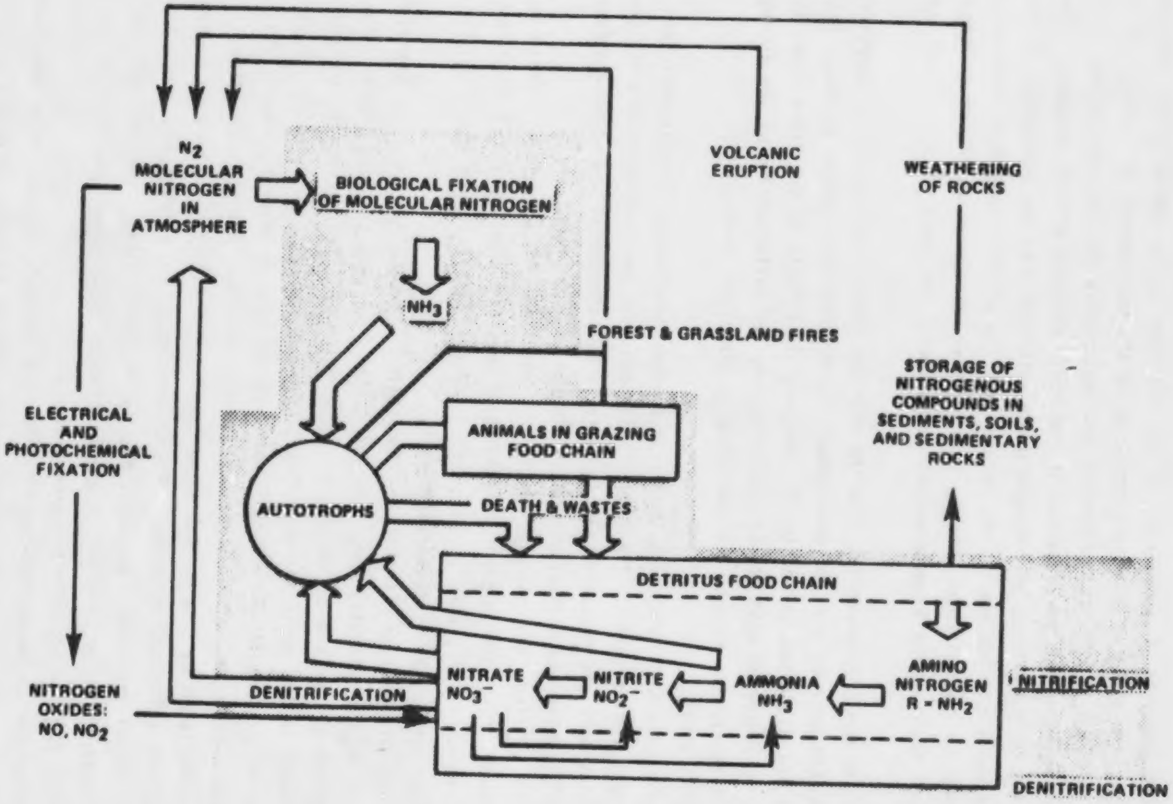
N_2
MOLECULAR NITROGEN IN ATMOSPHERE
BIOLOGICAL FIXATION OF MOLECULAR NITROGEN
NH_3
VOLCANIC ERUPTION
WEATHERING OF ROCKS
FOREST & GRASSLAND FIRES
STORAGE OF NITROGENOUS COMPOUNDS IN SEDIMENTS, SOILS, AND SEDIMENTARY ROCKS
ELECTRICAL AND PHOTOCHEMICAL FIXATION
ANIMALS IN GRAZING FOOD CHAIN
AUTOTROPHS
DEATH & WASTES
DETRITUS FOOD CHAIN
NITRATE NO_3^-
NITRITE NO_2^-
AMMONIA NH_3
AMINO NITROGEN $R - NH_2$
NITRIFICATION
NITROGEN OXIDES: NO, NO_2
DENITRIFICATION
DENITRIFICATION

Figure 4-1. The nitrogen cycle. Organic phase shaded.

are broken down through proteolysis to amino acids. The amino acids in turn are converted into ammonia (NH_3). The ammonia may be assimilated by aquatic or terrestrial plants and microorganisms may be bound by clay particles in the soil, or it may be converted by microorganisms to form nitrates in the process termed nitrification. It may also escape into the atmosphere.

4. Nitrification -- Nitrates are formed through the conversion by certain specific bacteria of ammonia first to nitrite (NO_2^-) and then to nitrate (NO_3^-). Nitrates may be assimilated by plants, washed downward through the soil into groundwater or through surface runoff into streams, rivers, and oceans, and may be transformed into atmospheric nitrogen or reduced to ammonia.

The organic phase of the nitrogen cycle is complete at this point where plants and microorganisms are able to assimilate the nitrates produced. Under certain circumstances nitrate reduction occurs; microorganisms may convert nitrates back to ammonia via the nitrate step. These processes are the converse of the previous transformations.

Nitrogen, as indicated above, once it is in the nitrate form may be lost from the soil in several ways. It may be assimilated by plants or microorganisms, or due to its solubility enter the soil solution and be carried off into the groundwater, lakes and streams, or through the process of denitrification converted into atmospheric nitrogen (N_2).

5. Denitrification -- Nitrates, through bacterial action, are converted into atmospheric nitrogen. Denitrification is an anaerobic process. Nitrates (NO_3^-) are converted into nitrites (NO_2^-), to nitrous oxide (N_2O) and finally into nitrogen gas (N_2) which goes off into the atmosphere. In the soil, nitrites rarely accumulate under acidic conditions, nitrites decompose spontaneously to nitric oxide (NO), and under alpine conditions, they are biologically converted to N_2O and N_2 (Alexander, 1977). It must be emphasized that this process is anaerobic and that conversion of nitrates to nitrites is extremely sensitive to the presence of atmospheric oxygen. If atmospheric oxygen is present, the conversion does not occur.

Denitrification is the greatest biological leak in the otherwise closed soil cycle (Alexander, 1977; Chapham, 1973). Through denitrification, nitrogen becomes unavailable to most plants and microorganisms and, therefore, also to animals. Nitrogen gas enters the large atmospheric reservoir wherein its residence time may be as great as 10^7 years (Delwiche, 1970). Nitrous oxide has a much shorter residence time. It may be converted to nitrogen gas in the stratosphere or returned by chemical or biological processes to the soil nitrogen pool. Were it not for biological nitrogen fixation, the molecular nitrogen released through denitrification would be lost to the organisms on earth.

Another pathway in the atmospheric phase of the nitrogen cycle is the oxidation of nitrogen by lightening to form nitrogen oxides. Nitrogen oxides in turn may react with water to form nitrates (Chapham, 1973).

4.3 THE GLOBAL CIRCULATION OF NITROGEN

The transformation and movement of nitrogen as explained in the foregoing pages related to the biogeochemical circulation of nitrogen. The circulation of nitrogen is a long term process. Global nitrogen resides in a number of different compartments or pools, the principal ones being primary rocks, sedimentary rocks, deep-sea sediments, the atmosphere, and the soil-water pool. The largest of these pools is the atmosphere composed of 79 percent molecular nitrogen (Bolin and Arrhenius, 1977; Delwiche, 1970, 1977). The atmosphere is not only an important reservoir for nitrogenous compounds but also serves as a principal conduit through which emissions of gaseous and particulate forms of the oxides of nitrogen (NO_x) are transformed and conveyed between terrestrial and aquatic systems. Söderlund and Svensson (1976) have hypothesized that a net flow of NO_x prevails from terrestrial to aquatic systems; losses of NO_x from aquatic system to the atmosphere were considered insignificant. Nitrogenous compounds occurring in the atmosphere can be returned to terrestrial or aquatic areas principally via wet (rainfall) or dry (particulate and gaseous) deposition (Chapter 6).

The distribution of nitrogen in the major compartments as estimated by Delwiche (1977) is listed in Table 4-1. Delwiche's figures are at variance with those of Söderlund and Svensson (1976) because of differences in compartment description; for example, "organic sea" as used by Delwiche includes an "active" 10 cm layer of the ocean bottom whereas Söderland and Svensson give 5.3×10^5 Tg for dissolved organic nitrogen. "Organic soil" is total whereas 3.0×10^5 Tg cited by the other authors is to a depth of one meter. In any case the figures are estimates and subject to change as new data are obtained.

Turnover times for the three largest "pools" of nitrogen are: 3×10^8 years for atmospheric nitrogen, 2,500 years for nitrogen in the seas when nitrates and organic compounds are counted together and less than one year for nitrates and nitrites in the soil (Whittaker, 1975). Delwiche (1970, 1977) has pointed out that the transfer rates can be estimated only within broad limits. The only two quantities of nitrogen known with any degree of accuracy are the amount of nitrogen in the atmosphere and the rate of industrial fixation (Delwiche, 1970). The amount and the length of time nitrogen is in the atmosphere indicates why the atmosphere is the greatest source of nitrogen, while the short period of time nitrogen is in the soil emphasizes why nitrogen is often in short supply as a nutrient element.

4.3.1 Important Nitrogen Fluxes

Various authors have estimated the global flow of nitrogen as it moves through the nitrogen cycle. The estimates of these authors are presented in Tables 4-2 and 4-3 for ease of comparison. Most of the estimates are based on extrapolation of experimentally-determined small-scale emission factors to the global scale, but some are crude estimates, arrived at by balancing mass flows to account for unknown sources. Particular reference will be made to those portions of the nitrogen cycle which are most influenced by human activities as it is in this context that the global flow of nitrogen becomes important.

TABLE 4-1. DISTRIBUTION OF NITROGEN IN MAJOR COMPARTMENTS

Compartment	Tg* N	Reference
Atmosphere	3.9×10^9	Garrels et al., 1975
Plants and animals	1.0×10^4	Delwiche, 1970
Organic, soil	1.7×10^5	Delwiche, 1970
Organic, sea	8.9×10^5	Delwiche, 1970
Inorganic, soil	1.6×10^5	Delwiche, 1970
Inorganic, sea	9.9×10^4	Delwiche, 1970
Sediments	2.0×10^8	Garrels et al., 1975

Delwiche, 1977

*Tg = 10^{12} grams

4.3.1.1 Biological Nitrogen Fixation--Biological nitrogen fixation involves the reduction of atmospheric N_2 to ammonia (NH_3), a form of fixed nitrogen which can be incorporated directly into the organic substances essential for life. Nitrogen fixation is important in the maintenance of soil fertility in terrestrial, aquatic, and agricultural ecosystems. It is indirectly responsible for the production of nitrates. Fixed forms of nitrogen serve as nutrient sources and, in certain circumstances involving high concentrations, are a source of environmental pollution.

It has been estimated by Burns and Hardy that, on a global basis, nitrogen fixation in terrestrial ecosystems accounts for 139 Tg (1 Tg = 10^{12}g) of fixed nitrogen produced each year; leguminous plants account for 35 Tg of this total with the remainder being produced in forests and grasslands (Burns and Hardy, 1975). Estimates of other investigators differ considerably and are shown in Tables 4-2 and 4-3.

Nitrogen fixation proceeds slowly in the presence of high levels of ammonia and other nitrogen-containing compounds, such as chemical fertilizer. Nitrous oxide (N_2O), a product of catabolic soil processes, has been shown in laboratory situations to have the potential for altering the rates of nitrogen fixation through inhibition of nitrogenase (Hardy and Knight, 1966).

In natural waters, the blue-green algae are the principal agents of nitrogen fixation. Fixation of nitrogen in aquatic regions of the world have been estimated by Soderlund and

TABLE 4-2. ESTIMATES OF GLOBAL NITROGEN FIXATION
IN THE BIOSPHERE
TgN/yr

	Delwiche (1970)	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)	Liu et al. (1977)	CAST (1976)
N_2-fixation (total)	92	245	224-324	166	227	N/A
terrestrial (biological)...	44	135	139	118	180	140
land and sea (biological)..	N/A	175	169-269	N/A	N/A	N/A
aquatic	10	40	30-130	N/A	37	0.36-3.6
combustion	18	20	19	16	18	21
other industrial	12	30	36	N/A	36	N/A
lightning (atmospheric) ...	7.6	10	N/A	N/A	9	N/A

TABLE 4-3. ESTIMATES OF GLOBAL EMISSIONS AND FLUXES OF OXIDES OF NITROGEN AND RELATED COMPOUNDS
TgN/yr

	Delwiche (1970)	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)	Liu et al. (1977)	Sze and Rice (1976)	CAST (1976)	Chameides et al. (1977)
Biological N_2-fixation (land and sea)...........	54	175	169-269	117	240	260	NA	NA
Biological denitrification (land and sea)...........	83 (N_2,N_2O)	190(N_2) 20(N_2O)	96-191(N_2) 36-149(N_2O)	338 (N_2O)	270 (N_2,N_2O)	260 (N_2,N_2O)	171-200 (N_2,N_2O)	NA
NO_x emissions from land to atmosphere............	NA	NA	40-108	NA	NA	NA	NA	NA
NO_x emissions from land and sea..................	NA	NA	NA	210(NO)	NA	NA	NA	NA
NO_x formed by combustion...	NA	15	19	15	NA	NA	NA	NA
NO_x formed by industrial processes...............	30	30	36	NA	40	NA	NA	NA
Atmospheric NH_3 transformation to NO_x.........	NA	30	3-8	NA	NA	NA	NA	NA
NH_3 emissions to atmosphere	NA	165(land and sea)	113-244 (land)	870(land and sea)	NA	NA	NA	NA
Atmospheric production of NO_x by lightning.........	NA	10	NA	NA	NA	NA	NA	30-40

Svensson (1976) to be in the range of 20 to 120 Tg N per year. The abundance of the blue-green algae, *Oscillatoria theibautii*, throughout oceanic areas, has been reported to account for a large percentage of the global nitrogen budget (Carpenter and Price, 1976).

4.3.1.2 Industrial Nitrogen Fixation--The need for fertilizers to be used in the growing of crops has resulted in the development of the Haber-Bosch process by which inorganically-based chemical fertilizers are produced. Soderlund and Svensson (1976) estimate that industrial fixation processes accounted for 36 Tg of fixed nitrogen produced in 1970, nearly 30 percent of the amount estimated from natural processes. These authors also estimate that combustion processes account for 19 Tg of NO_x-derived N emitted into the atmosphere in 1970. These processes release primarily NO.

4.3.1.3 Nitrogen Fixation by Other Processes--Minor amounts of nitrogen can be fixed in the atmosphere through chemical reactions. Molecular nitrogen can react with ozone (O_3) in stratospheric reactions to produce N_2O.

Lightning flashes in the troposphere can convert N_2 to NO via reaction with monatomic oxygen. Junge (1958) concluded from analysis of precipitation data that lightning can contribute only 10 to 20 percent of the amounts of nitrate found in rain. However, there are no direct experimental determinations of this estimate. Crutzen and Ehhalt (1977) estimated that from 8 to 40 Tg N are fixed each year by lightning.

4.3.1.4 Nitrates, Nitrites, and the Nitrogen Cycle--The microbial oxidation of NH_3 to nitrates (NO_3^-) and nitrites (NO_2^-) is the sole natural source of nitrate in the biosphere (Focht and Verstraete, 1977) other than atmospheric transformations of NO_x to nitrates (Chapter 6). Because nitrates are readily leached from soil and are susceptible to denitrification, nitrification can result in large losses of nitrogen from ammonium-based fertilizers applied to soils.

Nitrates are the fixed nitrogen forms predominant in stream and river effluents. In the atmosphere, transformation reactions involving NO, NO_2, NH_3 and acidic aerosols yield particulate nitrates, such as ammonium nitrate.

Nitrate is the predominant nitrogenous anion in atmospheric precipitation. Nitrite content is generally low (Georgii, 1963). The flux of NO_3^--N from wet deposition into terrestrial systems was estimated by Soderlund and Svensson (1976) to be in a range of 13 to 30 Tg NO_3^--N per year. Deposition in gaseous form was estimated by these authors to be in a range of 19 to 50 Tg NO_3^--N per year and 0.2 to 2.8 Tg NO_3^--N per year as particulates. By comparison, global river discharge of nitrate nitrogen (excluding polar and desert areas) was calculated at 8.1 Tg per year (Söderlund and Svensson, 1976). These estimates of nitrates and nitrite fluxes are summarized in Table 4-4.

4.3.1.5 Nitrates as Fertilizers--Man's greatest intervention into natural cycles has occurred because of the shortage of nitrogen as an available nutrient element in the soil (Delwiche, 1970). (It has been estimated that the amount of ammonia nitrogen (NH_4-N) that is converted

TABLE 4-4. ESTIMATES OF THE GLOBAL FLUX OF NITRATES AND NITRITES

TgN/yr

	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)
Atmospheric production from NO_2.......	20	N/A	95
Atmospheric production from NH_3.......	30	N/A	N/A
Total deposition (land and sea).......	60	18-51	95
Total dry deposition (land and sea)...	N/A	0.3-2.8	75
Total wet deposition (land and sea)...	N/A	18-46	20

by microorganisms to nitrate nitrogen (NO_3-N) is equivalent to the net nitrogen assimilation (0.017 kg/m^2) by plants each year.) (Bowen, 1966; Delwiche, 1977) To alleviate this shortage, the limiting factor in plant growth, industrial fixation of nitrogen was developed. At the present time the amount of nitrogen fixed industrially for the production of fertilizer equals the amount that was fixed by all terrestrial ecosystems before the advent of modern agriculture (Delwiche, 1970). The world's annual output of industrially fixed nitrogen was 30 million tons in 1968 and it has been estimated it will reach or exceed 100 million tons by the year 2000 (Parr, 1973). Consumption of fertilizer nitrogen in the U.S.A. will probably reach 11 million tons in 1980 (Parr, 1973). The impact of this environmental loading has not, until very recently, been considered.

4.4 AMMONIFICATION AND NITRIFICATION

Nitrogen fixation and denitrification processes in natural systems result in increased concentrations of ammonium ions (NH_4^+) in the soil or in aquatic systems as a result of ammonia (NH_3) hydration. These ions may be utilized by plants and bacteria to produce protein.

Ammonification is an important process in the renewal of the limited supply of inorganic nitrogen. Organic compounds, such as amino acids resulting from decay processes, are converted into NH_3 and ammonium ions. Volatilization of ammonia from soils may increase the atmospheric burden of NO_x as NH_3 undergoes atmospheric transformation (Chameides et al., 1977; National Research Council, 1978).

4.5 NITRIC OXIDE, NITROGEN DIOXIDE AND THE NITROGEN CYCLE

Total NO_x emissions to the atmosphere from terrestrial sources were reported by Söderlund and Svensson (1976) to be in the range of 8 to 25 Tg N per year. Estimates of other authors regarding NO_x emissions are presented in Tables 4-3 and 4-5.

TABLE 4-5. ESTIMATES OF THE GLOBAL FLUX OF NO_x
(NO AND NO_2)
TgN/yr

	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)	Crutzen and Ehhalt (1977)	Chameides et al. (1977)
Natural emissions from land to atmosphere........................	N/A	21-89	N/A	N/A	N/A
Natural emissions from land and sea to atmosphere.....................	N/A	N/A	210	N/A	N/A
Tropospheric production by lightning	10	N/A	N/A	8-40	30-40
Stratospheric production from N_2O...	5	0.3	2	N/A	N/A
Atmospheric production from NH_3.....	N/A	3-8	N/A	N/A	N/A
Production during combustion........	15	19	15	N/A	N/A
Other industrial production.........	30	36	N/A	N/A	N/A
Total land deposition...............	31	32-83	N/A	N/A	N/A
Total aquatic deposition............	18	11-33	N/A	N/A	N/A
Total wet deposition (as nitrates; land and sea)....................	49	18-46	75	N/A	N/A
Total dry deposition (land and sea).	11	25-70	151	N/A	N/A

While 40 to 108 Tg NO_x-N per year have been estimated to be released from terrestrial sources to the atmosphere, the bulk is reabsorbed and only 8 to 25 Tg NO_x-N escapes to the troposphere (Söderlund and Svensson, 1976). Hill (1971) has reported that NO and NO_2 are absorbed from the atmosphere by plants. Using data obtained from the experiments of Marakov (1969) and those of Kim (1973), Söderlund and Svensson (1976) estimate that soil contributes to the atmosphere between 1 to 14 Tg N in the form of NO and NO_2; losses from aquatic ecosystems to the atmosphere were considered by these authors to be minor. The principal source of gaseous NO_x in terrestrial systems is believed to be the chemical decomposition of nitrates (Söderlund and Svensson, 1976).

In the stratosphere, NO and NO_2 can be produced through photolysis and transformation reactions involving N_2O and O_3. Small amounts of these products (about 0.3 Tg annually) are expected to reenter the troposphere, mainly as NO_2 and HNO_3.

Tropospheric ammonia may be converted to NO, and indirectly to NO_2, via reaction with hydroxyl free radicals (·OH). Söderlund and Svensson (1976) estimate that this reaction accounts for 3 to 8 Tg NO_x-N produced annually. A higher estimate (20 to 40 Tg NO_x-N) was reported by Chameides and co-workers (1977).

Tropospheric production of NO_x during lightning discharges has been estimated to account for 8 to 40 Tg NO_x-N per year (Chameides et al., 1977; Griffing, 1977; Noxon, 1976).

The work of Chameides et al. (1977) suggests that lightning is a significant source of NO_x, producing about 30 to 40 Tg NO_x-N per year. If this estimate is correct, lightning could account for as much as 50 percent of the total atmospheric production of NO_x on a global basis (Chameides et al., 1977), or a level comparable to one estimate of the global average of man-made NO_x emissions (Crutzen et al., 1978; Noxon, 1978).

Direct observations by Noxon (1976) during a lightning storm indicate that ambient concentrations of NO_2 may be enhanced by a factor of 500 over the normal level. The enhanced levels decayed rapidly after passage of the storm. Liu et al. (1977) estimated NO_x production by lightning at 9 Tg N per year.

Recent studies of the global carbon cycle indicate that burning of wood and other biomass, largely in tropical land-clearing, may release significant quantities of NO_x (National Research Council, 1978).

Atmospheric NO_x returns to earth by two principal mechanisms: wet deposition (rain) and dry deposition (gaseous and particulate). Dry deposition has been estimated to be twice that of wet deposition over the same areas (Söderlund and Svensson, 1976).

Tropospheric reactions and transport considerations are discussed in more detail in Chapter 6.

4.6 NITROUS OXIDE AND THE NITROGEN CYCLE

Nitrous oxide is released from denitrification and nitrification processes in soils and aquatic environments and is transformed in stratospheric chemical reactions. It is not known to be involved in tropospheric reactions. After release from soil or water, N_2O diffuses to the stratosphere and may be converted to oxygen, nitrogen, and oxides of nitrogen (Junge, 1972). The stratosphere represents the only known atmospheric sink for N_2O.

From a review of the literature, Söderlund and Svensson (1976) estimated the N_2O flux from soils to the atmosphere in the range of 16 to 69 Tg N_2O-N per year. These authors also estimate that N_2O derived from denitrification processes account for at least 10 percent of the total amount of nitrogen denitrified. The authors caution, however, that the data available are too limited to draw more precise estimates. CAST (1976) estimated N_2O production in soils at 7 Tg N per year.

Total annual marine production and release to the atmosphere of N_2O was recently estimated at 4 to 10 Tg-N by Cohen and Gordon (1979). This estimate was based on new information which indicates major deficiencies in divergent earlier estimates by Hahn (1974) and Hahn and Junge (1977), who proposed a net source of 16 to 160 Tg-N/yr, and McElroy et al. (1976), who argued that the ocean could be a sink for 40 Tg-N/yr of atmospheric N_2O.

Söderlund and Svensson (1976), using data obtained by Junge (1972), estimate that 18 Tg N_2O-N per year is conveyed from the troposphere to the stratosphere and converted to N_2, O_3, and NO_x. These estimates are summarized in Table 4-6.

Nitrous oxide is important in the regulation of the amount of ozone in the stratosphere. Crutzen (1974) has suggested that an increase of 1 percent of the emission rate of nitrous oxide may cause a 0.2 percent decrease in stratospheric ozone, thus increasing the amount of ultraviolet radiation reaching the earth. The potential effect of coal- and gas-burning processes (Pierotti and Rasmussen, 1976) and the increased use of fertilizer nitrogen (Bremner and Blackner, 1978) on increasing N_2O levels has been suggested to be a cause of concern regarding O_3 destruction. Pierroti and Rasmussen (1976) report that coal-burning and gas-burning industrial processes may account for approximately 2 Tg N_2O-N produced each year. Liu and co-workers (1977) suggest that other land-based sources of N_2O could include sewage and nitrogen waste treatment plants.

Nitrous oxide has been reported to be released from soils during nitrification of added ammonium- or urea-based fertilizers under aerobic conditions (Bremner and Blackmer, 1978). Urea is rapidly hydrolyzed by a soil enzyme, urease, to form ammonium carbonate. Of the added nitrogen, approximately 0.04 percent was released as N_2O. Nitrous oxide also is discussed in relation to denitrification in Section 4.9.

4.7 ORGANIC NITROGEN AND THE NITROGEN CYCLE

Large transfers of organic nitrogen compounds are found within aquatic and terrestrial systems (Söderlund and Svensson, 1976). The nitrogen flow between these systems and the atmosphere is much less than the internal circulations.

Man's agricultural activities have been reported to be a cause of depletion of organic matter in soils (Paul, 1976; Stanford et al., 1975). Söderlund and Svensson (1976) have

TABLE 4-6. ESTIMATES OF THE GLOBAL FLUX OF NITROUS OXIDE
TgN/yr

	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)	Crutzen and Ehhalt (1977)	CAST (1976)
Natural emissions from land to atmosphere	13	16-69	N/A	12-80	7
Natural emissions from land and sea to atmosphere	20	N/A	342	N/A	N/A
Natural emissions from aquatic areas to atmosphere	7	20-80	N/A	40-90	N/A
Produced during atmospheric reaction of N_2 with ozone	15	N/A	N/A	N/A	N/A

estimated that 6 to 13 x 10^3 Tg-N are lost each year on a global basis (an order-of-magnitude-type estimate for agricultural lands only). Losses due to forest cutting were not taken into consideration.

Annual wet deposition of organic nitrogen has been estimated to be in a range of 10 to 100 Tg (Söderlund and Svensson, 1976). An average organic nitrogen content of 0.1 to 1.0 mg N per liter of terrestrial precipitation was used as the basis for the calculation. Organic nitrogen in river discharge was estimated at 9.9 Tg per year (Söderlund and Svensson, 1976).

4.8 AMMONIA AND THE NITROGEN CYCLE

There is considerable uncertainty as to the sources of atmospheric NH_3. A global source strength of 113 to 224 Tg-N per year has been estimated (Söderlund and Svensson, 1976), based on balancing of estimated total deposition. Sources such as excreta from wild and domestic animals and humans (Denmead et al., 1974; Healey et al., 1970; Luebs et al., 1973; Stanford et al., 1975) and coal combustion (Burns and Hardy, 1975; Georgii, 1963; Söderlund and Svensson, 1976) may account for a quarter of this total. Other likely major sources of NH_3 include decomposition of organic matter other than excreta, forest fires, and other wild fires, losses from manufacture, handling and application of ammonia-based fertilizers (Söderlund and Svensson, 1976) volatilization from oceans (Bouldin et al., 1974), and possibly from the senescing leaves of living plants (Fargahar et al., 1979). Estimation of atmospheric release from natural and agricultural areas is complicated by the observation that plant cover can reabsorb substantial amounts of the NH_3 released at ground level in these areas (Denmead et al., 1976).

It has been proposed that atmospheric NH_3 may be converted to NO_x by reaction with $\cdot OH$ radicals (McConnell, 1973; McConnell and McElroy, 1973). A recent estimate of the annual magnitude of this source is 20 to 40 Tg NO_x-N (Chameides et al., 1977). McConnell (1973) concluded that this reaction and heterogeneous losses are the dominant tropospheric NH_3 removal mechanisms. Atmospheric NH_3 returns to terrestrial and aquatic systems via precipitation (as ammonium salts), by dry deposition of gaseous NH_3 and particulate ammonium compounds (Söderlund and Svensson, 1976), and by direct gaseous uptake by plants (Denmead et al., 1976). Estimates of global flux of ammonia are summarized in Table 4-7.

4.9 DENITRIFICATION

In contrast to nitrogen fixation, denitrification results in the release of fixed forms of nitrogen, principally N_2O and N_2 with small amounts of NO, into the atmosphere. Ammonia production also has been reported (Payne, 1973; Stanford et al., 1975). As plants decay, a ubiquitous group of specialized microorganisms (e.g., Pseudomonas denitrificans) in soils and in aquatic systems reduce nitrates and nitrites to nitrogenous gases. Without these bacteria, most of the atmospheric nitrogen would be in the oceans or in sediments (Delwiche, 1970). On a time scale of millions of years, losses of these gases to the atmosphere are believed to be roughly balanced by the amounts of N_2 fixed by natural processes (Söderlund and Svensson, 1976). The ratio of N_2 to N_2O produced is an area of current interest because of the role of N_2O in the destruction of stratospheric ozone (Chapter 9).

Delwiche (1970) estimated that 83 Tg of N_2O-N is produced annually from terrestrial and oceanic sources. Estimates of other investigators of the extent of global denitrification are considerably higher than those of Delwiche. These estimates, as well as other estimates of the global budget for nitrogenous compounds, are presented in Tables 4-3 and 4-8. Soil factors such as acidity (Focht, 1974; Nommik, 1956), moisture content (Söderlund and Svensson, 1976), temperature (Nommik, 1956; Stanford et al., 1975), and oxygen content (Cady and Bartholomew, 1960; Nommik, 1956) determine proportions of NO, N_2O or N_2 produced. Major factors affecting the extent of denitrification losses in soils include availability of an organic carbon substrate (Focht, 1974; National Research Council, 1978), moisture content, presence or absence of a cover crop (Burford and Stefanson, 1973), and the type of fertilizer applied (Broadbent and Clark, 1965; Rolston et al., 1976).

Conditions favoring release of N_2O from nitrates include high soil acidity (low pH), presence of O_2, limited amounts of easily metabolizable organic compounds, low temperatures and high initial concentrations of nitrates. Acidity and temperature factors which increase N_2O production also tend to decrease the absolute rate of denitrification (Stanford et al., 1975). Molecular nitrogen is the principal end product under true anaerobic conditions (National Research Council, 1978). Nommik (1956) reported that NO production is favored in acidic anaerobic systems exposed to large amounts of nitrite. However, such conditions are not likely to occur in natural systems. Anaerobic conditions are promoted when soil moisture content increases, reducing the diffusion of oxygen through the soil pore spaces (Söderlund and Svensson, 1976). The production of N_2O by denitrification processes has been reviewed by CAST (1976), Hahn and Junge (1977), and Crutzen and Ehhalt (1977).

Denitrification also occurs in aquatic ecosystems, and the limited data available suggest that oceanic systems contribute greater amounts of N_2 and N_2O to the atmosphere than terrestrial systems (Söderlund and Svensson, 1976). Lake sediments are believed to provide ideal conditions for denitrifying organisms (Keeney et al., 1971; National Research Council, 1978).

4.10 ACIDIC PRECIPITATION

Increased amounts of NO_x released to the atmosphere is of concern due to their contribution to the acidification of precipitation (Likens, 1976). A full discussion pertaining to acidic precipitation appears in Chapter 11.

4.11 SUMMARY

Nitrogen is an element necessary for all life. The maintenance of an adequate balance among nitrogen-containing compounds is essential to the integrity of all ecosystems on earth. Nitrogen resides in five major reservoirs: primary rocks, sedimentary rocks, the deep-sea sediment, the atmosphere, and the soil-water pool. The web of pathways and fluxes by which oxides of nitrogen and associated nitrogenous compounds are produced, transformed, transported,

TABLE 4-7. ESTIMATES OF GLOBAL FLUX OF AMMONIA AND AMMONIUM COMPOUNDS
TgN/yr

	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Robinson and Robbins (1975)
Volatilized from land and sea......	165	N/A	860
Volatilized from land.............	N/A	113-244	N/A
Produced from burning of coal......	5	4-12	N/A
Terrestrial wet deposition.........	N/A	30-60	N/A
Wet deposition over oceans.........	N/A	8-25	N/A
Terrestrial dry deposition (gaseous)........................	N/A	57-114	679
Dry deposition over oceans (gaseous)........................	N/A	10-20	N/A
Precipitation as NH_4^+ (land and sea).........................	140	38-85	150
Dry deposition as NH_4^+ (land and sea).........................	N/A	3-8	N/A
Converted to NO_x in atmosphere.....	N/A	5-17	40

and stored in the principal nitrogen reservoirs, are commonly referred to as the nitrogen cycle.

An understanding of the nitrogen cycle is important in placing in perspective man's intervention as discussed in other chapters of this document.

The atmosphere, composed of 79 percent molecular nitrogen (N_2), is not only an important reservoir for nitrogenous compounds but also serves as a principal conduit through which emissions of gaseous and particulate forms of the oxides of nitrogen (NO_x) are transformed and conveyed between terrestrial and aquatic systems. Nitrogenous compounds occurring in the atmosphere can be returned to terrestrial or aquatic areas principally via wet (rainfall) or dry (particulate and gaseous) deposition.

Most estimates of the global flows of nitrogen compounds are based on extrapolation of experimentally-determined small-scale emission factors to the global scale; some are crude estimates, arrived at by balancing mass flows to account for unknown sources. Since published estimates differ greatly, it is difficult to assess with any certainty the fraction of NO_x

TABLE 4-8. ESTIMATES OF GLOBAL DENITRIFICATION
TgN/yr

	Delwiche (1970)	Burns and Hardy (1975)	Söderlund and Svensson (1976)	Sze and Rice (1976)	Liu et al. (1977)	CAST (1976)
Biological denitrification (total)..................	83	210	132-340	260	245	171-200
terrestrial..............	43	140	107-161	135	127	71-100
aquatic..................	40	70	25-179	125	118	100

emissions globally which arise from man's activities. In terms of their contribution to NO_x concentrations in polluted urban airsheds, however, it seems clear that natural processes are generally negligible (see Chapter 8).

The extent to which the use of industrially fixed nitrogen in agriculture has influenced the nitrogen cycle, the role of nitrous oxide in the nitrogen cycle, and the significance of increased amount of ammonia due to human activities are all matters of concern.

CHAPTER 4

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5. SOURCES AND EMISSIONS

5.1 INTRODUCTION

This chapter primarily reviews significant anthropogenic sources of nitrogenous compounds which directly affect human health or which may participate in atmospheric chemical pathways leading to effects on human health and welfare. Particular emphasis is placed on emissions of NO_x for two reasons: (1) NO_2 is a pollutant of major concern for human health (Chapter 15) and (2) atmospheric transformation products of NO_x such as nitric acid (HNO_3) and particulate nitrates are of concern both for their effects on human health and their role in the acidification of precipitation (Chapter 11). Emissions of nitrates, nitrites and nitrogenous acids are not discussed because, except in a few special situations (such as in the vicinity of nitric acid plants for HNO_3 or certain agricultural operations for particulate nitrates), they do not result in significant ambient concentrations. Atmospheric production and ambient concentrations of these compounds are discussed in Chapters 6 and 8, respectively. Agricultural usage of nitrogenous compounds is reviewed because of recent discussion of possible perturbations of the stratospheric ozone layer by nitrous oxide (N_2O) (Chapter 9), which is produced in part from fertilizer not utilized by plants. Sources of N-nitroso compounds and their possible precursors are reviewed as background material for a discussion of atmospheric burdens of these pollutants actually observed in ambient air (Chapter 8). Sources of ammonia are discussed because some researchers have suggested that it is converted to NO_x in the atmosphere.

5.2 ANTHROPOGENIC EMISSIONS OF NO_x

Global estimates of natural emissions of nitrogenous compounds including NO_x have been discussed in detail in Chapter 4. Since various authors differ greatly in their estimates of natural emissions, it is difficult to assess with any certainty the fraction of NO_x emitted globally from human activities.

In highly industrialized or populous localities, anthropogenic emissions of NO and/or NO_2 assume primary importance. Mobile combustion and fossil-fuel power generators are the two largest source categories. In addition, industrial processes and agricultural operations produce minor quantities. Although certain industrial processes, such as nitric acid plants, and certain agricultural activities, such as the application of fertilizer or the operation of animal feedlots, may result in localized emissions of other nitrogenous compounds, which have potential as NO_x sources through atmospheric transformation only NO and NO_2 are, in general, considered to be the primary pollutants; the other nitrogen oxides are mainly products of atmospheric reactions (Chapter 6).

In interpreting the emissions inventories to be presented throughout this chapter, it is important to note that NO_x emissions are generally calculated as though the compound being emitted were NO_2. This method of presentation serves the purpose of allowing ready comparison of different sources. Because of the interconvertibility of NO and NO_2 in photochemical smog reactions, such an approach has some merit and avoids the difficulty in interpretation associated with different ratios of NO/NO_2 being emitted by different sources. Two points, however, should be noted: (1) although NO is the dominant NO_x compound emitted by most sources, NO_2 fractions from sources do vary somewhat with source type and (2) conversion of NO_x emissions to NO_2 takes place over spatial and temporal scales which vary with the particular local circumstances of atmospheric pollutant mix, climatology and topography. For these reasons, NO_x emission inventories do not necessarily accurately reflect the potential of various sources for producing ambient NO_2 concentrations.

5.2.1 Global Sources of NO_x

Table 5-1 presents historical estimates of the man-made global production of NO_x (Robinson and Robbins, 1972,1975). These estimates are based on 1966 fuel consumption figures (U.S. Statistical Abstract, 1967) and emission factors available in 1965 (Mayer, 1965). From these data, Robinson and Robbins (1975) estimated global NO_x emissions from anthropogenic sources to be 48 x 10^6 metric tons per year (expressed as NO_2). These authors also estimated the ratio of natural emissions of NO_x from terrestrial and aquatic sources to those from anthropogenic sources to be about 7 to 1. An earlier estimate by Robinson and Robbins (1972) had set the ratio of natural emissions to anthropogenic emissions at 15 to 1. The downward revision was based on a 55 percent lower estimate of the amount of NO_2 emitted by natural sources.

TABLE 5-1. ESTIMATED ANNUAL GLOBAL EMISSIONS OF NITROGEN DIOXIDE (ANTHROPOGENIC)
(10^6 metric tons per year, expressed as NO_2)

Source	Emissions	Emissions as N	% Total
Total combustion and refining	48.0	14.6	100
Coal combustion	24.4	7.4	51
Petroleum refining	0.6	0.2	1
Gasoline combustion	6.8	2.1	14
Other oil combustion	12.8	3.9	27
Natural gas combustion	1.9	0.5	4
Other combustion	1.5	0.5	3

Source: Robinson and Robbins, 1975.

A different estimate was provided by Soderlund and Svensson (1976), who concluded that the ratio of NO_x emissions from natural sources to emissions from anthropogenic sources could range from roughly 1:1 to 4 or 5:1. It is clear that there are considerable uncertainties associated with global estimates of natural NO_x emissions (Chapter 4). In particular, estimates of different authors on the magnitude of NO_x emissions from soils vary greatly. Definitive experiments have yet to be performed.

5.2.2 Sources of NO_x in the United States

Table 5-2 and Figure 5-1 provide historical data on estimated emissions of NO_x in the United States for the years 1940 through 1970. A significant upward trend in the two major

TABLE 5-2. HISTORIC NATIONWIDE NO_x EMISSION ESTIMATES 1940-1970
(10^6 metric tons per year, expressed as NO_2)

Source Category	1940	1950	1960	1970
TRANSPORTATION	2.9	4.7	7.2	10.6
Motor vehicles	2.7	4.1	6.6	8.3
Aircraft	0	0	0	0.3
Railroads	0	0.2	0.1	0.1
Marine use	0.1	0.1	0.1	0.2
Nonhighway use	0.2	0.3	0.4	1.8
STATIONARY FUEL COMBUSTION	3.2	3.9	4.7	9.1
Electric utilities	0.5	1.1	2.1	4.3
Industrial combustion	1.8	1.8	1.6	4.1
Commercial	0.1	0.1	0.2	0.2
Residential	0.9	0.9	0.8	0.5
INDUSTRIAL PROCESS LOSSES	0	0.1	0.1	0.2
SOLID WASTE DISPOSAL	0.1	0.2	0.2	0.4
AGRICULTURAL BURNING	0.2	0.2	0.2	0.3
MISCELLANEOUS	0.7	0.4	0.2	0.1
Total	7.1	9.4	12.7	20.6

NOTE: A zero in this table indicates emissions of less than 50,000 metric tons/yr. Some totals do not agree due to rounding off.

Source: U.S. EPA, 1973.

source categories, transportation and stationary fuel combustion, is discernible over this 30-year period. Total NO_x emissions almost tripled. Recent emissions estimates, by year, for 1970 through 1976 are presented in Table 5-3 and Figure 5-2. Emissions from transportation sources increased by about 20 percent but emissions from stationary fuel combustion sources and total emissions did not exhibit monotonic behavior.

Minor discrepancies between Tables 5-2 and 5-3 for the year 1970 may be due to rounding errors and/or to changes in estimation methods used for producing the two tables. It is believed, however, that trends assessment is reliable within each table. The change in source category nomenclature is also to be noted.

Figure 5-3 shows the distribution of NO_x emissions by type of Air Quality Control Region (AQCR) for 1972. Large urban AQCR's, i.e. those having a Standard Metropolitan Statistical Area (SMSA) population exceeding 1,000,000, accounted for more than half (53 percent) of the NO_x emissions. The population in these same SMSA's was only 38.5 percent of the total U.S. population in 1970 (U.S. Bureau of the Census, 1973).

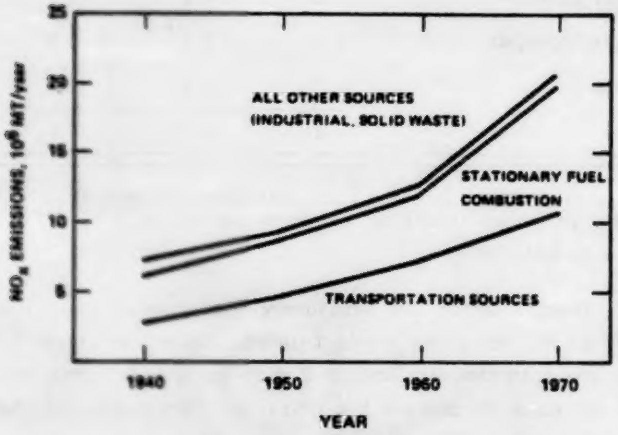


Figure 5-1. Historic NO_x emissions by source groups (U.S. EPA, 1973). (Values shown for each year are cumulative over source groups.)

TABLE 5-3. RECENT NATIONWIDE NO_x EMISSION ESTIMATES
(10^6 metric tons/yr, expressed as NO_2)

Source Category	1970	1971	1972	1973	1974	1975	1976
TRANSPORTATION	8.4	8.9	9.4	9.7	9.6	9.9	10.1
Highway vehicles	6.3	6.7	7.1	7.3	7.3	7.6	7.8
Nonhighway vehicles	2.1	2.2	2.3	2.4	2.3	2.3	2.3
STATIONARY FUEL COMBUSTION	10.9	11.2	11.7	12.1	11.9	11.2	11.8
Electric utilities	5.1	5.4	5.9	6.3	6.2	6.1	6.6
Industrial	5.1	5.1	5.1	5.1	5.0	4.5	4.5
Residential, commercial and institutional	0.7	0.7	0.7	0.7	0.7	0.6	0.7
INDUSTRIAL PROCESSES	0.6	0.6	0.7	0.7	0.7	0.7	0.6
Chemicals	0.2	0.2	0.3	0.3	0.3	0.3	0.3
Petroleum refining	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Metals	0	0	0	0	0	0	0
Mineral products	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Oil and gas production and marketing	0	0	0	0	0	0	0
Industrial organic solvent use	0	0	0	0	0	0	0
Other processes	0	0	0	0	0	0	0

(continued)

TABLE 5-3. (continued)

Source Category	1970	1971	1972	1973	1974	1975	1976
SOLID WASTE DISPOSAL	0.3	0.3	0.2	0.2	0.2	0.2	0.1
MISCELLANEOUS	0.2	0.3	0.2	0.2	0.2	0.2	0.3
Forest wildfires and managed burning	0.1	0.2	0.1	0.1	0.1	0.1	0.2
Agricultural burning	0	0	0	0	0	0	0
Coal refuse burning	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Structural fires	0	0	0	0	0	0	0
Miscellaneous organic solvent use	0	0	0	0	0	0	0
Totals	20.4	21.3	22.2	22.9	22.6	22.2	23.0

NOTE: A zero in this table indicates emissions of less than 50,000 metric tons/yr.

Source: U.S. EPA, 1977b.

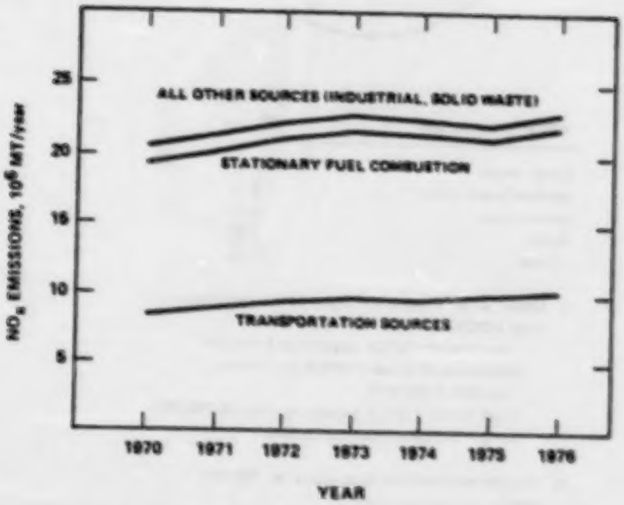
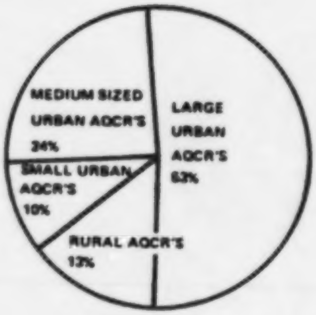


Figure 5-2. Recent NO_x emissions by source groups (U.S. EPA, 1976). (Values shown for each year are cumulative over source groups.)



AQCR Urbanization[a]	Emissions,[b] 10^6 tons/yr
Large Urban	11.71
Medium-sized Urban	5.30
Small Urban	2.36
Rural	2.88
Total	22.25

a. Urbanization is based on largest SMSA population in an AQCR:

Large Urban = SMSA population 1,000,000
Medium-sized Urban = SMSA population 250,000-1,000,000
Small Urban = SMSA population 50,000-250,000
Rural = AQCR containing no SMSA

b. Miscellaneous sources accounting for 160,000 tons/yr are not included.

Figure 5-3. Distribution of 1972 nationwide NO_x emissions by degree of urbanization (National Research Council, 1975).

A clear picture of the nationwide distribution of NO_x emissions can be obtained from the U.S. maps reproduced in Figures 5-4 through 5-6. Figure 5-4 shows total NO_x emissions by U.S. counties as compiled in the National Emissions Data System (NEDS) file of February 1978 (U.S. EPA, 1978). Regions of high source concentrations are evident near populous and industrial areas. When the total NO_x emissions per unit area (emission density) is plotted (Figure 5-5), the nationwide distribution tends to be more uniform than when emission totals alone are considered. Areas of relatively high emission densities are still evident throughout the eastern states and the midwest and on the west coast. The percent contribution of major point sources to total NO_x emissions is plotted by U.S. county in Figure 5-6. Comparison of this figure and Figure 5-4 reveals that, in general, major point sources make a significant contribution to total NO_x emissions in those areas where NO_x emissions are high. (In this discussion, a major point source is defined as one for which the yearly NO_x emissions exceed 100 tons.)

Table 5-4 documents NO/NO_x ratios in emissions from a variety of source types. Examination of the table reveals that NO is the dominant oxide of nitrogen emitted by most sources with NO_2 generally comprising less than 10 percent of the total NO_x emissions. It is interesting to note, however, that NO_2 forms upwards of 30 to 50 percent of the total NO_x emissions from certain diesel and jet turbine engines under specific load conditions. Tail gas from nitric acid plants, if uncontrolled, may contain about 50 percent NO_2. The variations in NO/NO_x ratios by source type reported in the table are possibly of some significance in local situations. An example might be in the immediate vicinity of a high-volume roadway carrying a significant number of diesel-powered vehicles. Situations of this type may assume increasing importance in the future because of the increasing interest in diesel-powered vehicles which generally have greater fuel economy than those with gasoline engines.

The national trends shown in Figures 5-1 and 5-2 do not reflect considerable local or regional differences in the relative amounts of NO_x emitted by the major source categories. For example, motor vehicles have been estimated to contribute approximately 90 percent of the NO_x emissions in Sacramento, California (California Board of Air Sanitation, 1966). In San Francisco, California, they are estimated to contribute about 56 percent (California Board of Air Sanitation, 1966), while in northwestern Indiana the estimate is 8 percent (Ozolins and Rehmann, 1968). Motor vehicle emissions in Los Angeles County, California, increased 6-fold from 1940 to 1970 (County of Los Angeles, 1971), compared to a 3-fold national increase.

While industrial process losses (NO_x emissions from noncombustion industrial sources) are minor on a national level, these emissions can be important near individual sources. Manufacturing of nitric acid, explosives and fertilizers, and petroleum refining are the principal activities in these source categories.

Aircraft are not considered a major source of NO_x on the national scale. Their impact in the immediate vicinity of major airports has been discussed recently (George et al., 1972; Jordan and Broderick, 1979). Although the total NO_x emissions associated with landing and takeoff operations at a large airport can be several thousand tons per year, most NO_x emissions

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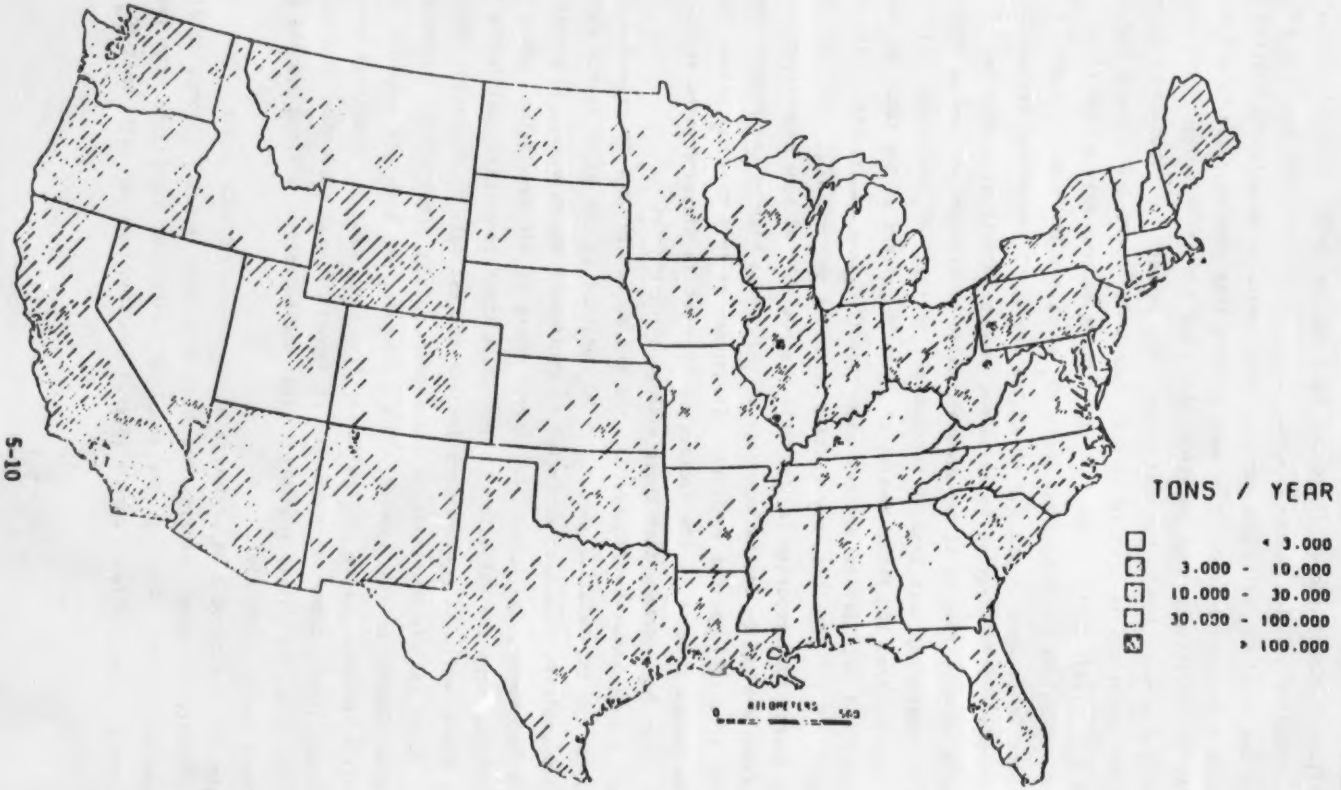
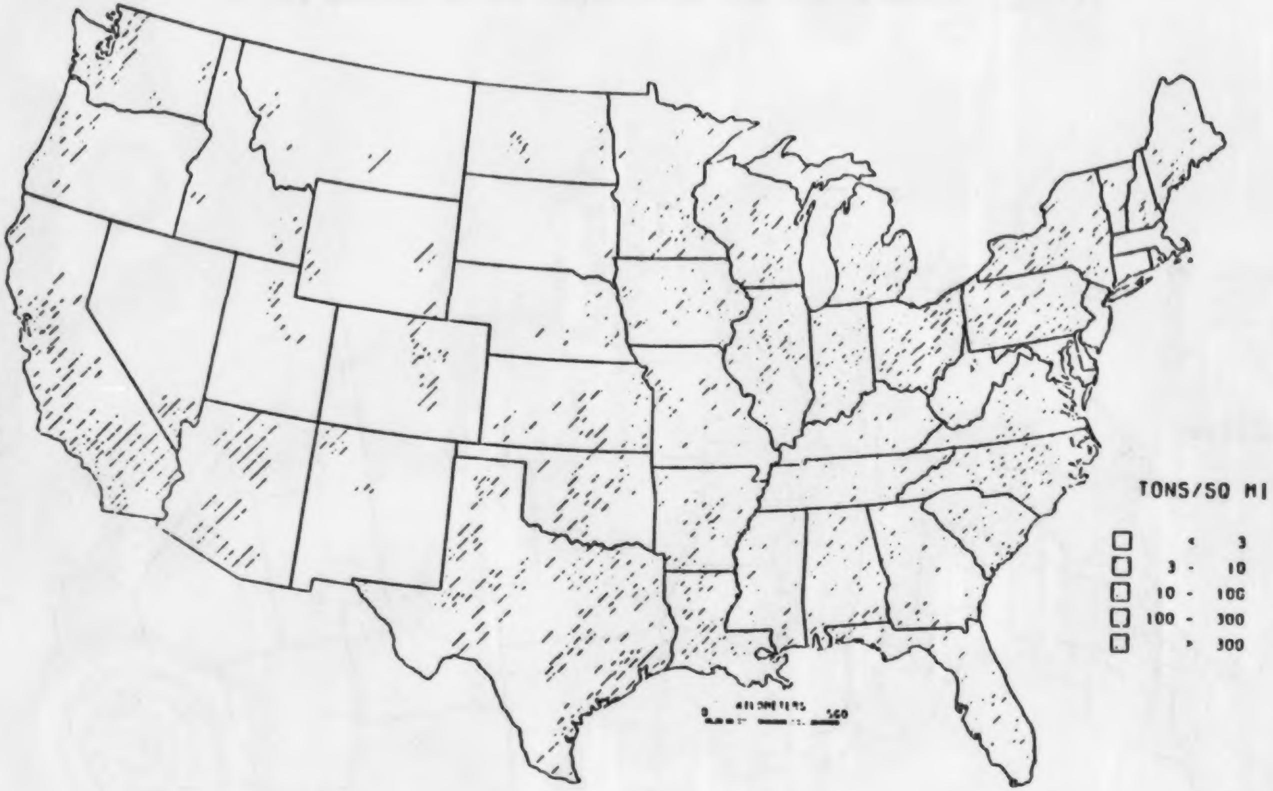

TONS / YEAR
< 3.000
3.000 - 10.000
10.000 - 30.000
30.000 - 100.000
> 100.000
KILOMETERS
0
500

Figure 5-4. Total NO_x emissions by U.S. county(U.S. EPA, 1978).


TONS/SQ MI
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3 - 10
10 - 100
100 - 300
> 300
KILOMETERS
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500

Figure 5-5. Total NO_x emission density by U.S. county (U.S. EPA, 1978).

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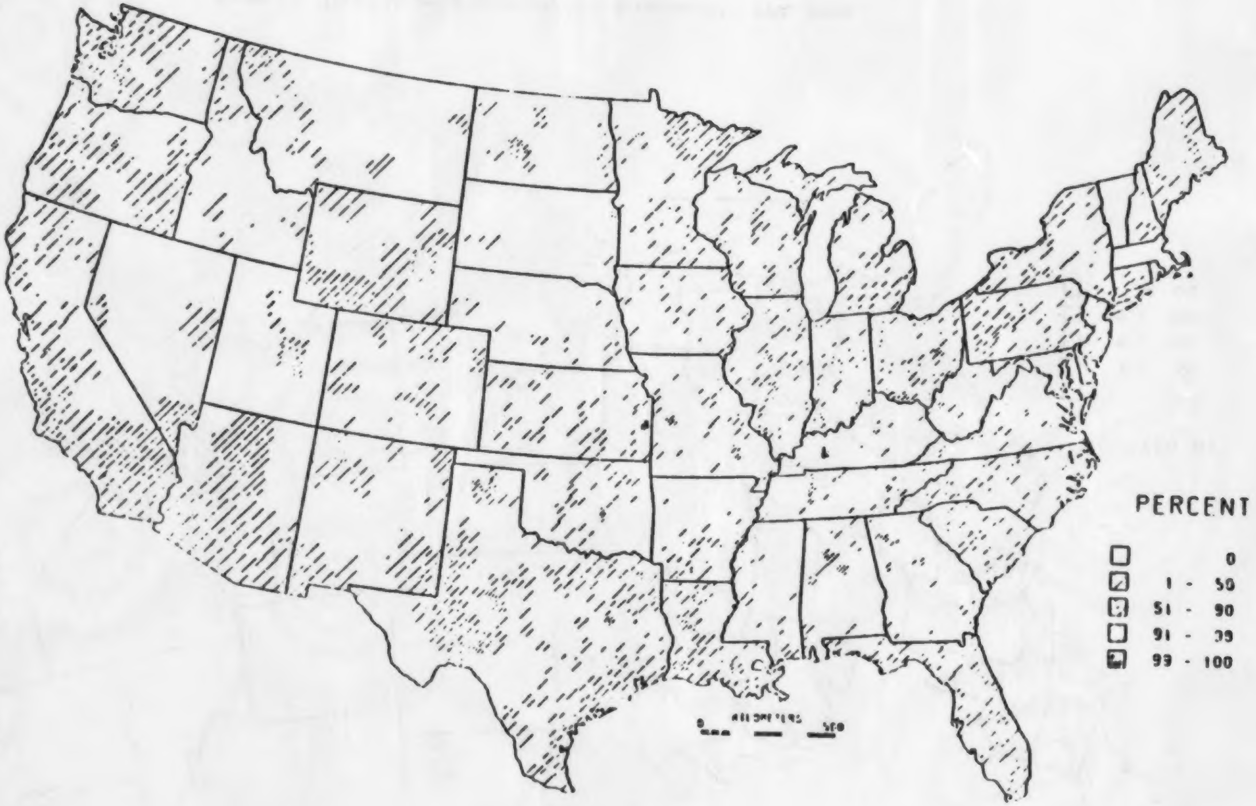
PERCENT
0
1 - 50
51 - 90
91 - 99
99 - 100
0 KILOMETERS 500

Figure 5-6. Percent NO_x emissions contributed by major point sources, by county (over 100 tons/yr) (U.S. EPA, 1978).

TABLE 5-4. NO/NO_x RATIOS IN EMISSIONS FROM VARIOUS SOURCE TYPES

Source Type	NO/NO_x	Reference
Uncontrolled tail-gas from nitric acid plants	~ 0.50	Gerstle and Peterson, 1966
Petroleum refinery heaters--using natural gas	0.93-1.00	Hunter et al., 1979
Linear ceramic tunnel kiln	0.90-1.00	
Rotary cement kilns	0.94-1.00	
Steel soaking pit--natural gas	0.97-0.99	
Wood/bark boiler	0.84-0.97	
Black liquor recovery boiler	0.91-1.00	
Carbon monoxide boiler	0.98-1.00	
Large 2-cycle internal combustion engine--natural gas	0.80-1.00	
Combined cycle gas turbine	0.83-0.99	
Gas turbine electrical generator--#2 fuel oil	0.55-1.00 (no load)-(full load)	Wasser, 1976
Industrial boilers--natural gas	0.90-1.00	Cato et al., 1976
Industrial boilers--#2 fuel oil	0.95-0.99	
Industrial boilers--PS 300 fuel	~ 0.96	
Industrial boilers--#6 fuel oil	0.96-1.00	
Industrial boilers--coal	0.95-1.00	
Industrial boilers--refinery gas	~ 0.95	
Industrial boilers--natural gas and #6 fuel oil	~ 1.00	
Industrial boilers--natural gas and refinery gas	~ 1.00	
Diesel-powered passenger car--Nissan	0.77-0.91 (idle)-(50mph)	Braddock and Bradow, 1975
Diesel-powered passenger car--Peugeot 204d	0.46-0.99 (idle)-(50mph)	Springer and Stahman, 1977a
Diesel-powered passenger car--various Mercedes	0.88-1.00	
Diesel-powered truck and bus--various engines	0.73-0.98	Springer and Stahman, 1977b
Mobile vehicles internal gasoline combustion engine	0.99-1.00	Wimmer and Reynolds, 1962 Campau and Neerman, 1966
Aircraft turbines (JT3D, TF30)	0.13-0.28(idle)[a] 0.73-0.92 (takeoff & cruise)	Souza and Daley, 1978

[a] Earlier studies (Lozano et al., 1968; Chase and Hurn, 1970) did not report such high idle concentrations of NO_2.

occur during approach, takeoff and climb out, distributing these emissions up to 3,000 feet above ground level and up to 10 miles from the airport. Even though high NO_2/NO_x ratios occur during idle of some aircraft turbines (Table 5-4), total emissions at idle are low. It is currently estimated that high-altitude emissions from existing and projected aircraft fleets do not represent a significant source of NO_x with respect to possible effects on stratospheric ozone, although substantial uncertainties remain (Broderick, 1978).

Factors influencing seasonal variabilities in mobile source NO_x emissions include temperature dependences of emissions per vehicle mile (about a 35 percent decrease in emissions for an ambient temperature increase from 20 to 90^oF) (Ashby et al., 1974), and changes in vehicle miles travelled seasonally (about 18 percent higher in summer than in winter, nationwide) (Federal Highway Administration, 1978). Further differences exist between vehicle miles travelled in urban and rural areas and among states in different parts of the country (Federal Highway Administration, 1978). Variations in NO_x emissions are also expected due to seasonal variation in power production from fossil fuel generating plants, which is estimated at 15 percent on a nationwide basis (U.S. Department of Energy, 1978). Production is greatest in the summer and least in the spring. Greater variations and different seasonal patterns have been reported for different areas of the country (California Board of Air Sanitation, 1966). Diurnal variations, notably those associated with motor vehicle traffic, are also important in their potential impact upon ambient air quality.

It should also be noted that the influence of the source categories considered in this section upon ambient concentrations at a given location depends upon factors such as land use, weather and climate, and topography. Emission estimates have other limitations as well. Aside from possible errors in establishing the emissions from each source, the spatial distribution of sources is usually not well known, since the inventories usually cover large areas. There is very little temporal resolution, which can often lead to poor understanding of the expected exposure. Many sources are intermittent. Representing such a source by annual emission data, therefore, underestimates the potential for short-term exposures. Also, meteorological parameters exhibit both seasonal and diurnal variability, which can greatly affect the impact of particular sources on ambient pollution levels.

5.3 EMISSIONS OF AMMONIA

On a global basis, abiotic emissions of ammonia (NH_3) represent only a small fraction of the total emissions of NH_3 (National Research Council, 1978; Robinson and Robbins, 1972; Soderlund and Svensson, 1976). Soderlund and Svensson (1976) calculated that anthropogenic emissions (from coal combustion) accounted for between 4×10^6 to 12×10^6 metric tons per year. A global loss of 7×10^6 metric tons NH_3-N per year from inefficiencies in handling and applying ammonia-based fertilizers was reported by the Council for Agricultural Science and Technology (CAST) (1976). A recent report (National Research Council, 1978) indicated that the United States accounts for about 25 percent of the global use of ammonia-based fertilizer. Another source (U.S. EPA, 1977a) has reported estimated NH_3 emissions for the United States at a much lower level, as shown in Table 5-5. Discrepancies in these estimates by various authors preclude any firm judgements at this time as to NH_3 emissions resulting from fertilizer usage.

TABLE 5-5. ESTIMATED AMMONIA EMISSION FROM FERTILIZER APPLICATION AND INDUSTRIAL CHEMICAL PRODUCTION IN U.S. (1975)

Source of Emission	Ammonia Emission Rate tons/yr	tons N/yr
Ammonia Production	19,000	15,600
Direct application of anhydrous ammonia[a]	168,000	138,400
Ammonium nitrate	59,000	48,600
Petroleum refineries	32,000	26,400
Sodium carbonate (Solvay process)	14,000	11,500
Diammonium phosphate	10,000	8,200
Ammoniator-granulators	10,000	8,200
Urea	4,000	3,300
Miscellaneous emission from fertilizer production	2,000	1,600
Beehive coke ovens	1,000	800
Total	319,000	262,600

[a] "Direct application" is the term used in agriculture when a chemical fertilizer is applied to the soil without combining or mixing it with any other chemical. Direct application of anhydrous ammonia involves transportation of ammonia to a storage area and to nurse tanks, metering, and injection into soil.

Source: (U.S. EPA, 1977a).

In addition to volatilization of NH_3 from use of fertilizers, emissions from feedlots may represent a significant local and regional source of ammonia. A National Research Council report on ammonia indicated that 50 to 100 percent of the urea-nitrogen in urine generated in feedlots may volatilize as ammonia (National Research Council, 1977). As urea is rapidly hydrolyzed into NH_3 and CO_2, atmospheric contributions of NH_3 from feedlot-generated urine of an estimated cattle population of 132 million in the United States could amount to 2×10^6 to 4×10^6 metric tons NH_3-N per year. This amount is between one-fourth and one-half of the rate of anthropogenic emissions of nitrogenous compounds (excluding N_2O) to the atmosphere in the United States (National Research Council, 1978).

5.4 AGRICULTURAL USAGE OF NITROGENOUS COMPOUNDS

Nitrogenous material applied as fertilizer participates in the nitrogen cycle via a variety of pathways (Chapters 4 and 12). Emissions of ammonia from agricultural sources have been discussed above in Section 5.3. Excess fertilizer may be of concern also: (a) by contributing an anthropogenically produced burden to stratospheric concentrations of N_2O with consequent potential for attenuation of the ozone layer (see Chapter 9) and (b) by contributing, through run-off from agricultural lands, to both nitrate pollution of drinking water and to changes in natural aquatic ecosystems. The data presented in Figure 5-7 and Table 5-6 are intended to place usage of nitrogenous materials applied as fertilizer in the U.S. in historical perspective. Examination of Table 5-6 reveals that the total nitrogen applied as fertilizer has increased more than a factor of 5 from 1955 to 1976. Applications of anhydrous ammonia have increased more than 14-fold and applications of nitrogen solutions have increased more than fifty times in the same period.

5.5 SOURCES OF N-NITROSO COMPOUNDS AND POSSIBLE PRECURSORS

N-nitroso compounds may be emitted to the atmosphere during their production or use, and have been postulated to occur by atmospheric formation or volatilization from water or soil. Additional routes of human exposure to N-nitroso compounds may include drinking water, foods and tobacco products.

5.5.1 Anthropogenic Sources of N-Nitroso Compounds

Possible direct anthropogenic sources of N-nitroso compounds include industrial processes in which these compounds are intermediate or final products, reactants or additives; or in which they may occur incidentally as impurities. While over 20 N-nitroso compounds are listed as products in recent commercial directories (Chem Sources, 1975,1977; Stanford Research Institute, 1977), only four of these were either sold in quantities over 1000 pounds yearly or resulted in annual sales over $1,000. Although the patent literature on N-nitroso compounds is sizeable, many are currently synthesized only for research purposes. The well-known toxic and carcinogenic effects of some N-nitroso compounds and related regulatory actions (Federal Register, 1974) have apparently discouraged their general use.

The two N-nitroso compounds produced in greatest quantity, diphenylnitrosamine and dinitrosopentamethylenetetramine, are used in the rubber industry as a vulcanizing retarder and a blowing agent, respectively (Magee, 1972). Neither has been found carcinogenic in laboratory tests on male rats (Boyland et al., 1968).

Several less direct sources of ambient N-nitroso compounds have been postulated. Some samples of cured meats, fish and fish meal, soya bean oil and tobacco have been shown to contain N-nitroso concentrations rarely exceeding 1.0 ppm (Fiddler, 1975; Ender et al., 1964; Hedler, 1968; Marquardt, 1971; Hoffman et al., 1974). Release of small quantities of N-nitroso compounds may occur in the processing of such products. Emissions occurring as a result of certain combustion processes have also been suspected. Analysis for emissions of nitrosamines (usually only N-nitrosodimethylamine) has been carried out as part of research programs

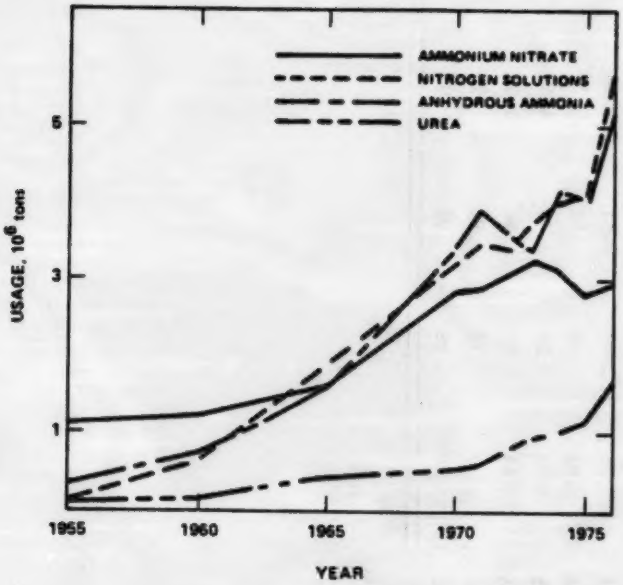

Figure 5-7. Trends in U.S. usage of nitrogenous material applied as fertilizer (Gerstle and Peterson, 1966).

TABLE 5-6. NITROGENOUS COMPOUNDS APPLIED AS FERTILIZER IN THE U.S. 1955-1976
(usage in 10^6 tons of material[a])

Material Applied	1955	1960	1965	1970	1973	1974	1975	1976
Total N	1.96	2.74	4.64	7.46	8.30	9.16	8.61	10.34
Ammonium Nitrate	1.12	1.23	1.63	2.84	3.28	3.17	2.81	2.94
Anhydrous Ammonia	.34	.71	1.56	3.47	3.41	4.18	4.02	4.91
Aqua Ammonia	.23	.43	.82	.70	.66	.72	.70	.68
Nitrogen Solutions	.11	.65	1.92	3.24	3.41	4.05	4.11	5.55
Urea	.07	.14	.43	.53	.96	1.03	1.15	1.62
Ammonium Sulfate	.52	.53	.77	.78	.95	.93	.82	1.04
Sodium Nitrate	.62	.45	.30	.09	.06	.05	.08	.06

[a] Numbers are rounded to two decimal places.

Source: HARGETT, 1977.

involving automobiles, diesel engines, emission control systems, and fuels and fuel additives. Tests by five different groups using different techniques all produced negative results (U.S. EPA, 1977e), although Fine et al. (1975) reported the presence of several unidentified N-nitroso compounds in the exhaust of a truck diesel engine and an automobile internal combustion engine. No firm explanation of these different results can be given at present.

5.5.2 Volatilization from Other Media

Some N-nitroso compounds, particularly those with low molecular weights, may reach the atmosphere through volatilization from soil and water. These compounds may be present in soil or water as deposits or effluents from industrial or agricultural sources, or may be formed de novo from appropriate precursors in water or soil. Nitrosamines have been found in several commercial herbicides (Fine and Ross, 1976) which, upon application, could be volatilized from surface water or soil. Volatilization may also occur from N-nitroso compounds formed in situ. Formation of N-nitroso compounds in soil and water samples has been demonstrated under laboratory conditions (Ayanaba and Alexander, 1974; Ayanabe et al., 1973; Elespuru and Lijinsky, 1973; Tate and Alexander, 1974; Wolfe et al., 1976). Most of these experiments involve pesticides or pesticide ingredients and nitrites, nitrates or nitrous acid, at levels which may reasonably be expected to occur in some farming or feedlot operations. Direct evidence is lacking, however, for the volatilization of N-nitroso compounds.

5.5.3 Atmospheric Formation: N-Nitroso Precursors

Possible mechanisms for atmospheric formation of N-nitroso compounds are reviewed in Chapter 6. In addition to the considerations presented in Chapter 6, it has been postulated that nitrosamines may be formed and emitted from industrial processes using amines to modify fly-ash resistivity (U.S. EPA, 1977e; U.S. EPA, 1975). A study, however, reported no detection of nitrosamines in emissions from a power plant using such amine additives (U.S. EPA, 1976).

In general, preliminary considerations of the atmospheric mechanisms suggested that ambient concentrations of nitrosamines should be investigated in the vicinity of emitters of two classes of airborne precursors: (1) oxides of nitrogen, nitrites, and/or nitrates and (2) amines, amides, or other related compounds (U.S. EPA, 1977e). Sources leading to ambient concentrations of the nitrogen compounds have been discussed above in Section 5.2. With regard to the second group, the only suspected N-nitroso precursors for which sources have been extensively documented are the amines. As of 1975, at least 32 companies were producing various amines (Chem Sources, 1975). Amines may be emitted directly from these production facilities, and also may be emitted during subsequent use in many manufacturing processes and products. Amines have been identified in emissions from decomposition of livestock and poultry manure, air sampled over cattle feedlots, and exhaust from rendering of animal matter (Peters and Blackwood, 1977; Shuval and Gruenar, 1972; U.S. EPA, 1977e). Volatility and water-solubility of various amines results in extensive dispersal of these compounds in the atmosphere, water and soil (Walker et al., 1976). Results of investigations of ambient concentrations of N-nitroso compounds in the vicinity of suspected sources are discussed in Section 8.3. These investigations have failed to detect evidence of atmospheric N-nitroso formation.

5.5.4 N-Nitrosamines in Food, Water and Tobacco Products

This section reviews briefly the major non-atmospheric routes of human exposure to N-nitroso compounds in order to place possible exposure via the atmospheric route in perspective.

A variety of raw and processed foods have been tested for N-nitroso compounds. A review of earlier qualitative studies was published by Sebranek and Cassens (1973). Scanlon (1975) has summarized a number of the studies reported since 1970. These recent studies concentrate on processed meats and fish, in which nitrosamines, nitrosopiperidine and nitrosopyrrolidine have been identified at levels usually considerably less than 1 ppm. Raw and cooked bacon samples were found by several investigators to contain 1.5 to 139 ppb nitrosopyrrolidine (NPY) and 1.0 to 30 ppb dimethylnitrosamine (DMN) (Allison et al., 1972; Crosby et al., 1972; Fazio et al., 1973; Fiddler et al., 1974; Havery et al., 1976; Pensabene et al., 1974; Sen et al., 1973a,1974). Other meat products (including luncheon meat, frankfurters and other sausages) were found to contain 3 to 105 ppb NPY, 1 to 94 ppb DMN, 2 to 25 ppb diethylnitrosamine (DEN) and 50 to 60 ppb nitrosopiperidine (NPi) (Allison et al., 1972; Fazio et al., 1972; Panalaks et al., 1973,1974; Sen, 1972; Sen et al., 1973b; Wasserman et al., 1972). Raw and processed fish products have shown 1 to 26 ppb DMN and 1 to 6 ppb NPY (Allison et al., 1972; Crosby et al., 1972; Fazio et al., 1971), with some fish meal samples containing as much as 450 ppb DMN (Sen et al., 1972). Cheese samples were found to contain up to 4 ppb DMN, 1.5 ppb DEN and 1.0 ppb NPY (Allison et al., 1972; Crosby et al., 1972). Negative results have been obtained in other tests for nitrosamines on many food samples, including bacon, ham and other pork products, fats and oils, cheeses, and total diet samples (Havery et al., 1976).

Contamination of drinking water with N-nitroso coumpounds may occur through water-borne emissions from the industrial sources discussed in Section 5.5.1 or by nitrosation of precursors found in natural bodies of water or water supply and treatment systems (U.S. EPA, 1977c,1977d,1977e). N-nitroso compounds have been found in industrial wastewater, and in samples taken from water near industrial facilities. Nitrosation in lake water samples has been demonstrated in the laboratory (Ayanaba and Alexander, 1974). Analyses of drinking water have either failed to detect N-nitroso compounds or have shown concentrations in the 0.1 ppb range (Fine et al., 1975a,1975b).

There may be significant exposure to N-nitroso compounds in use of cigarettes and other tobacco products. Mainstream smoke of blended unfiltered U.S. cigarettes was found to contain the following N-nitroso compounds (amounts in nanograms per cigarette): nitrosodimethylamine (84), nitrosoethylmethylamine (30), nitrosonornicotine (137), and nitrosodiethylamine (<5) (Hoffman et al., 1975). N'-nitrosonornicotine found in a variety of chewing tobacco products indicates that such unsmoked products may also be sources of exposure to N-nitroso compounds (Hoffman et al., 1974).

5.6 SUMMARY

5.6.1 Sources and Emissions of NO_x

Oxides of nitrogen have their origin in a number of natural (Chapter 4) and anthropogenic processes. Since various authors differ greatly in their estimates of natural emissions, it is difficult to assess with any certainty the fraction of total global NO_x emissions originating from human activities. In terms of sources giving rise to significant human exposure, however, the most important emissions occur as a result of man's burning of fossil fuels such as coal, oil or gasoline. Mobile combustion and fossil-fuel stationary combustion are the two largest source categories, comprising about 44 percent and 51 percent of the nationwide anthropogenic NO_x emissions, respectively, 1976. In the stationary combustion category, electric utilities were responsible for approximately 56 percent of the anthropogenic NO_x emissions, and industrial combustion accounted for another 38 percent. In the mobile combustion category, highway vehicle emissions comprised about 77 percent, with the rest attributable to non-highway vehicles. In addition, non-combustion industrial processes such as petroleum refining and manufacture of nitric acid, explosives and fertilizers may produce quantities of NO_x which, while minor on a national basis, may be significant near individual sources.

In most ambient situations nitrogen dioxide (NO_2), the NO_x compound of most concern for human health and welfare, is not emitted directly into the atmosphere in significant amounts (typically less than 5 percent of NO_x emissions occur as NO_2). It arises mainly from the oxidation in the atmosphere of the more commonly emitted compound nitric oxide (NO). However, some minor source types, notably a number of diesel and jet turbine engines do, under certain load conditions, emit a significant fraction of their NO_x emissions as NO_2.

In general, the relationship between the magnitude of NO_x emissions and resulting ambient NO_2 concentrations is neither direct nor constant. The influence of sources upon ambient concentrations at a given location depends not only on details of the emissions from the source but also upon such factors as the presence or absence of other atmospheric constituents such as hydrocarbons and ozone, as well as land use, weather and climate, and topography.

Estimates of historical emissions for the years 1940 through 1970 indicate that total anthropogenic emissions of NO_x almost tripled during that period. More recently, emissions from transportation sources increased by about 20 percent from 1970 to 1976, but emissions from stationary combustion sources and total emissions did not exhibit a monotonic upward trend. Examination of NO_x emissions inventories by U.S. county reveals that, in general, both point and area sources contribute significantly in those places where total NO_x emissions are high. There are, however, considerable local or regional differences in the relative amounts of NO_x emitted by the major source categories. For example, motor vehicles have been estimated to contribute approximately 90 percent of the NO_x emissions in Sacramento, California, while the corresponding statistic in northwestern Indiana is only 8 percent. Emissions may also exhibit a significant diurnal and/or seasonal variability. The seasonal variation in power production from fossil fuel generating plants, e.g., is about 15 percent on a nationwide basis. Diurnal variations in traffic volume usually lead to marked daily emission profiles for mobile sources. Representing such sources by annual emissions data only may underestimate their potential for producing high short-term concentrations. Meteorological variables also exhibit both seasonal and diurnal variability, which can greatly affect the impact of particular sources on ambient pollution levels.

5.6.2 Sources and Emissions of Other Nitrogenous Compounds

Principal anthropogenic sources of ammonia (which some researchers have suggested is converted to NO_x in the atmosphere) include coal combustion, inefficiencies in handling and applying ammonia-based fertilizers, and volatilization of the urea-nitrogen in animal urine generated in feedlots. The last source has been estimated to contribute between one-fourth and one-half of all the anthropogenically produced nitrogenous compounds emitted yearly to the atmosphere in the United States (N_2O is not included in this estimate).

The use of nitrogen-based fertilizer (which some researchers have implicated in increased N_2O emissions leading to possible depletion of the stratospheric ozone layer) has risen markedly in the last two decades in the United States. The total nitrogen applied as fertilizer has increased by more than a factor of 5 from 1955 to 1976. Applications of anhydrous ammonia have increased more than 14-fold and applications of nitrogen solutions have increased more than 50 times during the same period. However, recent estimates of the effects of increased N_2O on stratospheric ozone indicate a much smaller effect than previously suspected (Chapter 9).

Possible direct atmospheric sources of N-nitroso compounds (some of which are known carcinogens) include industrial processes in which these compounds are intermediate or final products, reactants or additives, or may occur incidentally as impurities. Other than NO_x, the only proposed N-nitroso precursors for which sources have been extensively documented are the amines. Amines have been identified in emissions from decomposition of livestock and poultry manure, air sampled over cattle feedlots, and exhaust from rendering of animal matter. There is, however, little or no evidence to date to indicate that the atmospheric route for human exposure to nitrosamines is a cause for concern (Chapter 8).

CHAPTER 5

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6. ENVIRONMENTAL TRANSPORT AND TRANSFORMATION

This chapter is an assessment of the atmospheric behavior of the oxides of nitrogen. It is concerned with the processes and mechanisms that govern the dispersion and geographical movement of the oxides of nitrogen from their sources, the chemical and physical transformations that may occur within the atmosphere or in removal processes, atmospheric residence times, and removal mechanisms.

The predominant form of the oxides of nitrogen emitted to the atmosphere from man-made sources is nitric oxide. In the atmosphere, nitric oxide is converted chemically to a number of secondary products, including nitrogen dioxide, nitrites, nitrates, and nitrosamines. In addition, nitric oxide emissions contribute chemically to ozone formation. The chemical transformation of the oxides of nitrogen to these secondary products occurs simultaneously with transport and removal. The object of this chapter is to provide a brief survey of the transformation and transport of nitrogen oxides. Appropriate references are provided for further detail. Section 6.1 is devoted to the chemistry of the oxides of nitrogen in the lower atmosphere. The reactions involving oxides of nitrogen are first summarized and discussed. Then, laboratory evidence on the relationship between NO_2 levels and precursors is cited. Chemical reactions occurring in plumes and computer simulation of atmosphere chemistry are discussed. The formation of nitrites and nitrates is surveyed in Section 6.2. Section 6.3 discusses the transport and removal of nitrogenous species and currently available techniques for predicting atmospheric NO_2 concentrations when sources of NO_x are known (source-receptor relations). Section 6.4 is devoted to the chemistry of nitrosamine formation.

6.1 CHEMISTRY OF THE OXIDES OF NITROGEN IN THE LOWER ATMOSPHERE

Solar radiation triggers a series of reactions in the atmosphere between gaseous organic molecules and nitrogen oxides, producing a wide variety of secondary pollutants. The totality of primary and secondary pollutants involved in these photochemical reactions is known as photochemical smog. To understand the chemistry of the oxides of nitrogen in the lower atmosphere, it is necessary to consider the interactions that take place between the oxides of nitrogen and organic constituents. Several reviews of atmospheric chemistry are available (Heiklen, 1976; Seinfeld, 1975; Stern, 1977), as are detailed discussions of reaction mechanisms (Baldwin et al., 1977; Carter et al., 1978; Demerjian et al., 1974; Falls and Seinfeld, 1978; Whitten and Hogo, 1977) and rate constants (Hampson and Garvin, 1975). In this section the chemistry of the oxides of nitrogen in the lower atmosphere is briefly reviewed. The above-cited references should be consulted for more detail.

Most of the chemistry that occurs in a sunlight-irradiated urban atmosphere involves the interaction of a variety of unstable, excited molecules and molecular fragments that have only a transitory existence. These species include: the unexcited and first excited electronic states of the oxygen atom, triplet-P oxygen atoms [$O(^3P)$], and singlet-D oxygen atoms [$O(^1D)$]; ozone (O_3); symmetrical nitrogen trioxide (NO_3); dinitrogen pentoxide (N_2O_5); hydroxyl radicals

(HO); alkylperoxyl radicals (RO_2); acylperoxyl radicals $RC(O)O_2$; and other less important species. In the formulas, R represents a methyl (CH_3), ethyl (C_2H_3), or another, more complex hydrocarbon radical. The paths by which these intermediates are formed and destroyed are important keys in explaining the chemical changes that occur in the polluted atmosphere.

6.1.1. Reactions Involving Oxides of Nitrogen

The major portion of the total oxides of nitrogen emitted by combustion sources is nitric oxide (NO). The rate at which NO is converted to nitrogen dioxide (NO_2) through oxidation by the oxygen in air:

$$2NO + O_2 \rightarrow 2NO_2 \tag{6-1}$$

is proportional to the square of the nitric oxide concentration since two molecules of NO are required for the oxidation; it is, therefore, very sensitive to changes in nitric oxide concentration. Reaction 6-1 can be important in the vicinity of sources in generating up to 25 percent of total NO_x during the initial state of dilution with air when the concentration of NO is still quite high. Reaction 6-1 is much too slow, however, to account for any significant fraction of the nitric oxide to nitrogen dioxide conversion in the atmosphere for typical ambient levels of nitric oxide.

Since sunlight triggers the phenomenon of photochemical smog formation, it is important to recognize those constituents that will absorb light energy. In some cases, these constituents decompose or become activated for reaction. A dominant sunlight absorber in the urban atmosphere is the brown gas, nitrogen dioxide. Light absorption at wavelengths <430 nm can cause the rupture of one of the nitrogen-oxygen (N-O) bonds in the nitrogen dioxide (O-N-O) molecule and generate the reactive ground state oxygen atom, the triplet-P oxygen atom, and a nitric oxide molecule. The efficiency of this process is wavelength-dependent:

$$NO_2 + \text{sunlight (290-430 nm)} \rightarrow O(^3P) + NO \tag{6-2}$$

The highly reactive triplet-P oxygen atom formed in air collides frequently with oxygen molecules. During such encounters ozone may be formed:

$$O(^3P) + O_2 + M \rightarrow O_3 + M \tag{6-3}$$

M in this equation represents a nitrogen, oxygen, or other third molecule that absorbs the excess vibrational energy released, thereby stabilizing the ozone produced. For most concentration conditions common in polluted atmospheres, the very reactive ozone molecules regenerate nitrogen dioxide by reaction with nitric oxide:

$$O_3 + NO \rightarrow NO_2 + O_2 \tag{6-4}$$

Alternatively, ozone may react with nitrogen dioxide to create a new transient species, symmetrical nitrogen trioxide:

$$O_3 + NO_2 \rightarrow NO_3 + O_2 \tag{6-5}$$

The nitrate species forms dinitrogen pentoxide, the reactive anhydride of nitric acid, by reaction with nitrogen dioxide:

$$NO_3 + NO_2 \rightarrow N_2O_5 \tag{6-6}$$

Dinitrogen pentoxide may redissociate to form symmetrical nitrogen trioxide and nitrogen dioxide or possibly react with water to form nitric acid ($HONO_2$):

$$N_2O_5 \rightarrow NO_3 + NO_2 \qquad (6\text{-}7)$$

or

$$N_2O_5 + H_2O \rightarrow 2HONO_2 \qquad (6\text{-}8)$$

The following reactions may take place between oxygen atoms and NO_2 and NO:

$$NO_2 + O(^3P) \rightarrow NO + O_2 \qquad (6\text{-}9)$$

or

$$NO_2 + O(^3P) + M \rightarrow NO_3 + M \qquad (610)$$

or

$$NO + O(^3P) + M \rightarrow NO_2 + M \qquad (6\text{-}11)$$

Also, NO and NO_3 may react to regenerate NO_2:

$$NO_3 + NO \rightarrow 2NO_2 \qquad (6\text{-}12)$$

Nitrous acid is produced by:

$$NO + NO_2 + H_2O \rightarrow 2HONO \qquad (6\text{-}13)$$

and may react bimolecularly to regenerate the original reactants:

$$HONO + HONO \rightarrow NO + NO_2 + H_2O \qquad (6\text{-}14)$$

The unexcited and first excited electronic state of the oxygen atom are produced by ozone photolysis in sunlight:

$$O_3 + \text{sunlight} \begin{cases} \text{290-350 nm} \rightarrow O_2 + O(^1D) \text{ or } O(^3P) & (6\text{-}15) \\ \text{450-700 nm} \rightarrow O_2 + O(^3P) & (6\text{-}15b) \end{cases}$$

The singlet-D oxygen $[O(^1D)]$ atom is much more reactive than the ground state triplet-P oxygen $[O(^3P)]$ atom. For example, it reacts efficiently during collision with a water molecule to form an important transient species in the atmosphere, the hydroxyl radical:

$$O(^1D) + H_2O \rightarrow 2HO \qquad (6\text{-}16)$$

This radical is also formed through the photodecomposition of nitrous acid (HONO):

$$HONO + \text{sunlight (290-400 nm)} \rightarrow HO + NO \qquad (6\text{-}17)$$

The hydroxyl radical can reassociate with nitrogen dioxide to produce nitric acid:

$$HO + NO_2 + M \rightarrow HONO_2 + M \qquad (6\text{-}18)$$

or form nitrous acid by reacting with nitric oxide:

$$HO + NO + M \rightarrow HONO + M \qquad (6\text{-}19)$$

A careful review of the net results of reactions 6-1 through 6-19 reveals that these reactions alone cannot explain the rapid conversion of NO to NO_2 observed in the ambient atmosphere. In fact, if these reactions alone occurred, the original supply of nitrogen dioxide in our atmosphere would be slightly depleted under irradiation with sunlight, and a small and near constant level of ozone would be created in a few minutes. The key to the observed nitric oxide to nitrogen dioxide conversion lies in a sequence of reactions between the transient species present and other reactive molecules such as the hydrocarbons and aldehydes present in the polluted atmosphere.

In the presence of hydrocarbons the number of reactions greatly increases. Thus, the hydroxyl radicals produced by reactions 6-16 and 6-17 can react with a hydrocarbon (paraffin, olefin, aromatic, or any compound having C-H bonds):

$$OH + \text{Hydrocarbon} \rightarrow R\cdot + H_2O \qquad (6\text{-}20)$$

Reaction 6-20 produces an alkyl radical (R·) which contains a free electron. This radical quickly picks up an oxygen molecule from the air to form a peroxyl radical $RO_2\cdot$:

$$R\cdot + O_2 \xrightarrow{M} RO_2\cdot \qquad (6\text{-}21)$$

Typically, the next reaction in the series converts NO to NO_2 and produces an oxyl radical, RO·:

$$RO_2\cdot + NO \rightarrow RO\cdot + NO_2 \qquad (6\text{-}22)$$

A hydrogen abstraction by molecular oxygen may then produce a hydroperoxyl radical, $HO_2\cdot$. The rest of such an RO· radical typically forms a carbonyl compound, OHC:

$$RO\cdot + O_2 \rightarrow OHC + HO_2\cdot \qquad (6\text{-}23)$$

Finally, the hydroperoxyl radical (HO_2) can react with a second NO to form NO_2 to complete the cycle:

$$HO_2\cdot + NO \rightarrow OH\cdot + NO_2 \qquad (6\text{-}24)$$

Although this description is very simplified, these series of reactions contain the essential features of NO to NO_2 oxidation and subsequent ozone formation.

The initial source of radicals is very important; although the rate and yield of oxidant formation depend on many other factors, the length of the induction period before accumulation of oxidant depends strongly on the initial concentration of radicals. (The length of the induction period is important primarily in constant light intensity smog chambers. Diurnally varying radiation tends to lessen the importance considerably.) In smog chambers and possibly in the ambient atmosphere, the photolysis of nitrous acid, reaction 6-17, may be the most important initial source of radicals. Nitrous acid has been detected in smog chambers in concentrations sufficient to explain the observed induction time for smog chemistry, but the concentrations necessary to initiate smog chemistry in the atmosphere are below the limits measured by most modern instruments.

Another possible source of radicals in the atmosphere is the photolysis of aldehydes:

$$RCHO + h\nu \rightarrow H\dot{C}O + R\cdot \qquad (6\text{-}25)$$

Aldehydes are emitted from many sources, including automobiles. They are also formed in smog.

During the course of the overall smog formation process, the free radical pool is maintained by several sources, but the dominant one appears to be photolysis of the aldehydes formed from the initial hydrocarbons. Since the reactions of free radicals with NO form a cyclic process, any additional source of radicals will add to the pool and increase the cycle rate. Conversely, any reaction that removes free radicals will slow the cycle rate. For example, a primary radical sink and a primary sink for oxides of nitrogen is reaction 6-18 to form nitric acid.

The hydrocarbon classes important in the chemistry of the polluted troposphere are alkanes, olefins, and aromatics. In addition, the oxygenated hydrocarbons, such as aldehydes, ketones, esters, ethers, and alcohol are also important. A great variety of chemical reactions take place among these organic species and the free radicals cited above. The reactions of typical hydrocarbon species are now discussed briefly. Throughout the discussion references to more extensive coverages are given.

The most important atmospheric reaction involving alkanes is with the OH radical. For n-butane, for example, the reaction is

$$CH_3CH_2CH_2CH_3 + OH\cdot \rightarrow CH_3CH_2CH_2CH_2\cdot + H_2O \qquad (6\text{-}26a)$$

and

$$\rightarrow CH_3CH_2\dot{C}HCH_3 + H_2O \qquad (6\text{-}26b)$$

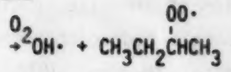
The alkyl radicals will rapidly add O_2 to form the corresponding peroxyalkyl radicals, e.g.

$$CH_3CH_2CH_2CH_2\cdot + O_2 + M \rightarrow CH_3CH_2CH_2CH_2O_2\cdot + M \qquad (6\text{-}27)$$

(subsequently the third body M will not be indicated). A reaction of substantially lesser importance is with oxygen atoms,

$$CH_3CH_2CH_2CH_3 + O(^3P) \xrightarrow{O_2} OH\cdot + CH_3CH_2CH_2CH_2O_2\cdot \qquad (6\text{-}28a)$$

and

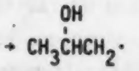


$$\xrightarrow{O_2} OH\cdot + CH_3CH_2\overset{OO\cdot}{\overset{|}{C}}HCH_3 \qquad (6\text{-}28b)$$

The importance of both the OH and $O(^3P)$ reactions with alkanes is the generation of the peroxyalkyl radical $RO_2\cdot$, which plays a substantial role in the conversion of NO to NO_2. Rate constants for alkane reactions are summarized by Hampson and Garvin (1975).

The atmospheric chemical reactions involving olefins have been widely studied (Carter et al., 1978; Demerjian et al., 1974; Niki, 1978). The most important reactions in which olefins participate are with OH radicals, ozone, and atomic oxygen, in that order. The reaction of OH with an olefin, such as propylene, may proceed by addition of OH to the double bond or by abstraction of a H-atom from the olefin. For propylene, for example, the reaction paths with OH are:

$$CH_3CH = CH_2 + OH\cdot \rightarrow CH_3\dot{C}HCH_2OH \qquad (6\text{-}29a)$$

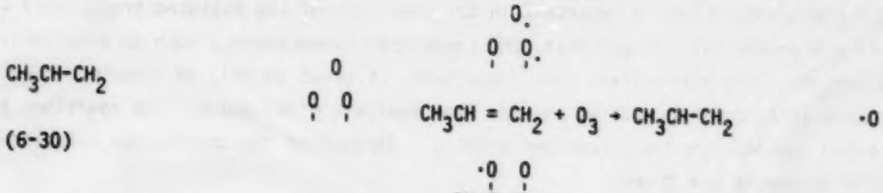


$$\rightarrow CH_3\overset{OH}{\overset{|}{\dot{C}}}HCH_2\cdot \qquad (6\text{-}29b)$$

$$\rightarrow CH_2\dot{C}H = CH_2 + H_2O \qquad (6\text{-}29c)$$

In each case the free radical product will quickly react with O_2 to produce a peroxyalkyl radical that is capable of converting NO to NO_2.

Ozone-olefin reactions are a source of free radicals and stable products in air pollution chemistry. The initial attack of O_3 on an olefin produces an unstable intermediate, which may decompose by several pathways (Niki, 1978; O'Neal and Blumstein, 1973). For propylene, for example, the initial step in the reaction with O_3 is believed to be:



$$CH_3CH = CH_2 + O_3 \rightarrow CH_3CH-CH_2 \qquad (6\text{-}30)$$

Subsequent composition of the products leads to a variety of free radicals and stable products (Herron and Huie, 1977; Niki et al., 1977). The mechanisms of ozone-olefin reactions are still under considerable study, although most of the potential paths have been delineated.

The reaction of olefins with atomic oxygen plays a minor role in olefin consumption and radical and product formation. Again, for propylene the reaction is:

$$CH_3CH = CH_2 + O(^3P) \rightarrow CH_3CH_2\cdot + H\dot{C}O \qquad (6\text{-}31a)$$

or

$$\rightarrow CH_3\dot{C}O + CH_3\cdot \qquad (6\text{-}31b)$$

or

$$\rightarrow CH_3CH_2CHO \qquad (6\text{-}31c)$$

The mechanism of photooxidation of aromatic species in the atmosphere is perhaps the area of greatest uncertainty in atmospheric hydrocarbon chemistry. The principal reaction of aromatics is with the hydroxyl radical (Hendry, 1978; Perry et al., 1977). For aromatic-OH reactions, the initial step can be either addition to or abstraction from the aromatic ring (Kenley et al., 1978). The free radical addition products may then react, most likely with either O_2 or NO_2, leading to the cresols or nitrotoluenes, respectively. The abstraction route probably leads to benzaldehyde. The mechanism of aromatic-OH reactions is yet to be clarified.

Aldehydes, both aliphatic and aromatic, occur as primary and secondary pollutants and are direct precursors of free radicals in the atmosphere (Lloyd, 1978). Consequently, aldehyde chemistry represents an important subject area in atmospheric chemistry. Although aldehydes are the main oxygenated hydrocarbons generally considered with respect to their role in atmospheric chemistry, other classes of oxygenated hydrocarbons, such as ketones, esters, ethers and alcohols, are present and participate to a somewhat lesser extent. Major secondary sources of aldehydes include the reactions of ozone and OH radicals with hydrocarbons, and radical decomposition products. In addition, aromatic aldehydes can be formed as an ultimate consequence of the reaction of OH with aromatics, e.g. benzaldehyde. The aldehydic hydrogen-carbon bond in aldehydes is relatively weak (CH bond strength is 86 kcal/mol^{-1}). Consequently, this hydrogen atom will be susceptible under atmospheric conditions to attack by radical species, such as O(^{3}P), O(^{1}D), OH, and HO_2. Of these OH is by far the most dominant. Hydroxyl radicals are generally thought to abstract a H-atom from aldehydes--chiefly the aldehydic H-atoms, i.e.

$$OH + RCHO \rightarrow H_2O + RCO \qquad (6\text{-}32)$$

If one assumes an atmospheric concentration of 10^6 radicals cm^{-3}, the rates of decay of HCHO and CH_3CHO by reaction with OH are approximately 4.2 percent and 5.8 percent per hour, respectively (Lloyd, 1978).

The photodissociation of aldehydes is an important radical generation mechanism in the formation of photochemical air pollution. The reactions that are most significant can be generalized in terms of a radical and a molecular route:

$$RCHO + h\nu \rightarrow R + H\dot{C}O \qquad (6\text{-}33a)$$
and
$$\rightarrow RH + CO \qquad (6\text{-}33b)$$

(Reaction 6-33a was previously given as reaction 6-25.) The radical route is the more important one from the point of view of atmospheric chemistry.

As hydrocarbons are oxidized in photochemical smog, they generally produce formaldehyde, HCHO, at some point. Considerable attention has been given to formaldehyde photolysis in recent years. There appears to be general agreement that the primary paths are:

$$HCHO + h\nu \rightarrow H\cdot + HCO \qquad (6\text{-}34a)$$
and
$$\rightarrow H_2 + CO \qquad (6\text{-}34b)$$

Moortgat and Warneck (1979) recently measured the quantum yield for the photolysis of formaldehyde. Their results imply that the radical pathway (reaction 6-34a) and the nonradical pathway (reaction 6-34b) occur at approximately equal rates under typical atmospheric conditions.

Another primary reaction path of formaldehyde in photochemical smog is the reaction with OH·:

$$HCHO + OH\cdot \longrightarrow H_2O + HCO \qquad (A)$$

In order to compare the rates of photolysis with the depletion of formaldehyde by OH reaction, one can calculate a photolysis rate of approximately 13 percent per hour for a solar zenith angle of 20° using the value of the photodissociation rate given by Horowitz and Calvert (1978).

Formaldehyde photolysis represents an important source of radicals in smog chemistry through reaction of the products with O_2 yielding the hydroperoxyl radical, $HO_2\cdot$. Thus:

$$H\cdot + O_2 + M \longrightarrow HO_2\cdot + M \qquad (B)$$
$$HCO\cdot + O_2 \longrightarrow HO_2\cdot + CO \qquad (C)$$

Since reactions B and C are very fast in normal atmospheres, reactions 6-34a and 6-34b are frequently written as:

$$HCHO + h\nu \xrightarrow{2O_2} 2\,HO_2\cdot + CO \qquad (D)$$
$$\text{and } HCHO + OH\cdot \xrightarrow{O_2} H_2O + HO_2\cdot + CO \qquad (E)$$

A rate constant for reaction E was recently recommended by the NASA Panel for Data Evaluation (NASA, 1979).

Acetaldehyde, CH_3CHO, also appears to play a significant role in smog chemistry. In this case, photolysis proceeds mainly by the radical pathway: (Calvert and Pitts, 1966).

$$CH_3CHO + h\nu \longrightarrow CH_3 + HCO\cdot \qquad (E)$$

Another primary reaction is the abstraction of hydrogen by the OH radical:

$$CH_3CHO + OH\cdot \longrightarrow CH_3\ C(O)\cdot + H_2O \qquad (G)$$

Subsequent reactions of the product radicals of reactions E and G with atmospheric O_2 are very fast so that one may write:

$$CH_3CHO + h\nu \xrightarrow{2O_2} CH_3O_2 + HO_2 + CO \qquad (H)$$

$$\text{and } CH_3CHO + OH\cdot \xrightarrow{O_2} CH_3C(O)O_2\cdot + H_2O \qquad (I)$$

Acetaldehyde chemistry thus introduces the chemistry of alkylperoxy radicals (RO_2) via the methylperoxy radical, CH_3O_2; and the chemistry of peroxyacyl nitrates [$RC(O)O_2NO_2$] via the formation of peroxyacetyl nitrate (PAN) from the acetylperoxy radical, $CH_3(CO)O_2$ (see reactions 6-42 ff below).

In addition to aldehydes, the ketones, methyethylketone and acetone are known to play a role in photochemical smog (Demerjian et al., 1974).

The interaction with NO and NO_2 of the organic free radicals produced by hydrocarbon oxidation represents an extremely important aspect of the chemistry of the oxides of nitrogen in the polluted atmosphere. The radicals can be classed according to:

$R\cdot$	alkyl	$R\overset{O}{\overset{\|}{C}}\cdot$	acyl
$RO\cdot$	alkoxyl		
$ROO\cdot$	peroxyalkyl	$R\overset{O}{\overset{\|}{C}}O\cdot$	acylate
		$R\overset{O}{\overset{\|}{C}}OO\cdot$	peroxyacyl

In air it can be assumed that combination with O_2 is the sole fate of alkyl ($R\cdot$) and acyl ($RCO\cdot$) radicals and that the reaction is essentially instantaneous. Consequently, in reactions with alkyl or acyl radicals as products, these products are often written as the corresponding peroxy radicals. Also, acylate radicals will decompose rapidly to give an alkyl radical and CO_2. Therefore, only alkoxyl, peroxyalkyl, and peroxyacyl radicals need to be considered explicitly in terms of NO_x chemistry. Table 6-1 shows the various reaction combinations that are important between these radicals and NO and NO_2.

The reactions of OH with NO_2 and NO are reasonably well understood and have been previously listed as reactions 6-18 and 6-19. Rate constants for these two reactions are available (Hampson and Garvin, 1975).

The rate constant for the reaction of HO_2 and NO has recently been determined by direct means and is substantially larger than previously calculated indirectly (Howard and Evenson,

TABLE 6-1. REACTIONS OF ALKOXYL, ALKYLPEROXYL AND ACYLPEROXYL RADICALS WITH NO AND NO_2

	NO		NO_2	
Free Radical	Reaction	Reference	Reaction	Reference
OH	$OH + NO \rightarrow HONO$	Hampson and Garvin, 1978	$OH + NO_2 \rightarrow HONO_2$	Tsang et al., 1977
HO_2	$HO_2 + NO \rightarrow NO_2 + OH$	Howard and Evenson, 1977	$HO_2 + NO_2 \rightarrow HONO + O_2$ $\rightarrow HO_2NO_2$ $(HO_2NO_2 \rightarrow HO_2 + NO_2)$	Howard, 1977[20] Graham et al., 1977
RO	$RO + NO \rightarrow RONO$ $(RONO + h\nu \rightarrow RO + NO)$	Batt et al., 1977	$RO + NO_2 \rightarrow RONO_2$ $\rightarrow RCHO + HONO$	Wiebe et al., 1973
RO_2	$RO_2 + NO \rightarrow NO_2 + RO$ $\rightarrow RONO_2$		$RO_2 + NO_2 \rightarrow RO_2NO_2$ $(RO_2NO_2 \rightarrow RO_2 + NO_2)$	
RCO_3	$RCO_3 + NO \rightarrow NO_2 + RCO_2$	Cox and Roffey, 1977 Hendry and Kenley, 1977	$RCO_3 + NO_2 \rightarrow RCO_3NO_2$ $(RCO_3NO_2 \rightarrow RCO_3 + NO_2)$	Cox and Roffey, 1977 Hendry and Kenley, 1977

1977). The HO_2-NO reaction, as noted earlier, is a key reaction in the atmospheric conversion of NO to NO_2.

The reaction of HO_2 and NO_2 has the following two possible mechanisms (Howard, 1977). Reaction 6-35b is not considered to be important in atmospheric chemistry:

$$HO_2 + NO_2 \rightarrow HO_2NO_2 \qquad (6\text{-}35a)$$

and

$$HO_2 + NO_2 \rightarrow HONO + O_2 \qquad (6\text{-}35b)$$

In addition, the peroxynitric acid formed in reaction 6-35a thermally decomposes as follows (Graham et al., 1977):

$$HO_2NO_2 \rightarrow HO_2 + NO_2 \qquad (6\text{-}36)$$

At the present time it appears that, at the temperatures prevalent in summer smog episodes (>20°C), peroxynitric acid does not represent an appreciable sink for NO_2 because of the rapid thermal decomposition reaction 6-36. At lower temperatures HO_2NO_2 will achieve higher concentrations and its importance as a sink for NO_2 increases.

The reactions of RO, RO_2 and RCO_3 with NO and NO_2 represent key reactions in the conversion of NO to NO_2 and the formation of organic nitrites and nitrates.

The main alkoxyl radical reactions with NO and NO_2 are:

$$RO\cdot + NO \rightarrow RONO \qquad (6\text{-}37a)$$

or

$$\rightarrow RCHO + HNO \qquad (6\text{-}37b)$$

and

$$RO\cdot + NO_2 \rightarrow RONO_2 \qquad (6\text{-}38c)$$

or

$$\rightarrow RCHO + HONO \qquad (6\text{-}38b)$$

The reaction of alkylperoxyl radicals with NO is generally assumed to proceed by the oxidation of NO to NO_2 with formation of an alkoxyl radical:

$$RO_2\cdot + NO \rightarrow NO_2 + RO\cdot \qquad (6\text{-}22)$$

Reaction 6-22 is believed to be an important route for the oxidation of NO to NO_2 in the atmosphere (the alkoxyl radical may react further to produce HO_2, which also converts NO to NO_2).

It has been postulated that longer chain peroxyalkyl radicals (n>4) from alkane photooxidation will add to NO to form an excited complex that can be stabilized to produce an alkyl nitrate (Darnall et al., 1976):

$$RO_2\cdot + NO \rightarrow RONO_2 \qquad (6\text{-}39)$$

The peroxyalkyl-NO_2 reaction proceeds principally by

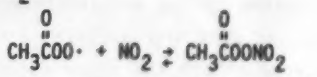
$$RO_2\cdot + NO_2 \rightarrow RO_2NO_2 \qquad (6\text{-}40)$$

The peroxynitrate may thermally decompose according to

$$RO_2NO_2 \rightarrow RO_2\cdot + NO_2 \qquad (6\text{-}41)$$

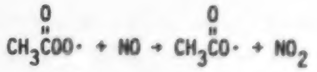
Measured rate constants for the RO_2-NO_2 reaction and the RO_2NO_2 decomposition are not currently available.

Peroxyacyl nitrates have long been recognized as important components of photochemical air pollution (U.S. EPA, 1978). Peroxyacetyl nitrate (PAN) exists in equilibrium with the peroxyacyl radical and NO_2:



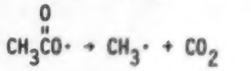
$$CH_3\overset{O}{\overset{\|}{C}}OO\cdot + NO_2 \rightleftarrows CH_3\overset{O}{\overset{\|}{C}}OONO_2 \qquad (6\text{-}42)$$

There exists a competition between NO and NO_2 for the peroxyacyl radical through:



$$CH_3\overset{O}{\overset{\|}{C}}OO\cdot + NO \rightarrow CH_3\overset{O}{\overset{\|}{C}}O\cdot + NO_2 \qquad (6\text{-}43)$$

The acetyl radical will rapidly decompose as follows:



$$CH_3\overset{O}{\overset{\|}{C}}O\cdot \rightarrow CH_3\cdot + CO_2 \qquad (6\text{-}44)$$

followed by:

$$CH_3\cdot + O_2 \rightarrow CH_3O_2\cdot \qquad (6\text{-}21)$$

$$CH_3O_2\cdot + NO \rightarrow CH_3O\cdot + NO_2 \qquad (6\text{-}22)$$

$$CH_3O\cdot + O_2 \rightarrow HCHO + HO_2\cdot \qquad (6\text{-}23)$$

$$HO_2\rightarrow + NO \rightarrow OH\cdot + NO_2 \qquad (6\text{-}24)$$

Thus, PAN chemistry is intimately interwoven in the NO to NO_2 conversion process. Rate constants for reactions 6-42 and 6-43 have recently been reported by two groups of investigators (Cox and Roffey, 1977; Hendry and Kenley, 1977).

The chemistry of the oxides of nitrogen in a hydrocarbon-containing atmosphere can be summarized as follows: the major observed phenomenon in the system is conversion of NO to NO_2 and formation of a variety of nitrogen-containing species, such as nitrites and nitrates. The conversion of NO to NO_2 is accompanied by accumulation of O_3. NO_2 serves as both an initiator and terminator of the chain reactions that result in conversion of NO to NO_2 and buildup of O_3. Termination of the chain reactions leads to nitric acid and organic nitrates. The nature of the system can be explained by considering its behavior as a function of the initial concentrations of NO_x and hydrocarbon in the irradiation of a static system, as well as the ratio of two reactants, i.e, the $[HC]/[NO_x]$ ratio.

At low $[HC]/[NO_x]$ ratios (usually ratios of less than about 1 to 2/1) the rate at which NO is converted to NO_2 is influenced by the availability of organic compounds. Therefore, the effects of reducing organic compounds are to slow the conversion of NO to NO_2, thereby lowering the NO_2/NO ratio. When this occurs, a larger proportion of the NO that is converted to NO_2 occurs through the destruction of ozone. This then has to the overall effect of reducing the rate of ozone formation. If the oxidation of NO by organics is delayed sufficiently so that the sun has passed its zenith before significant amounts of NO_2 are created, photodissociation of NO_2 will be diminished and less ozone will accumulate on that day. At moderately high

[HC]/[NO_x] ratios (usually greater than about 5 to 8/1), the greater availability of organic radicals means that all of these radicals are not consumed as rapidly in reactions with NO, and more reactions between the radicals and NO_2 are able to occur. Thus, the amount of ozone formed and accumulated begins to become limited by the availability of NO_x, and becomes less sensitive to additional organic precursors. At very high [HC]/[NO_x] ratios (greater than about 20 to 30/1), ozone cannot accumulate because either the ozone is consumed by reaction with hydrocarbons or radical-radical termination reactions occur which reduce oxygen atom and, hence, ultimate ozone concentration.

Identification of the nitrogen-containing products in atmospheric reactions has been under investigation for a number of years (Gay and Bufalini, 1971; Pitts, 1977; Spicer and Miller, 1976). In general, the most important gaseous nitrogen-containing products in the NO_x-organic system are nitric acid and PAN. As noted, reactions of NO and NO_2 with free radicals produce, in addition to nitrous, nitric, and peroxynitric acids, a variety of organic nitrogen-containing species (Table 6-1). There currently exist important areas of uncertainty with regard to the formation of nitrogen-containing products in atmospheric reactions. The extent of formation and decomposition of peroxynitrates, RO_2NO_2, is unknown, and rate constants for the key reactions in the series, RO_2 + NO, are yet to be determined.

6.1.2 Laboratory Evidence of the NO_2-to-Precursor Relationship

In the previous section, the nature of chemical reactions involving oxides of nitrogen and hydrocarbons in the atmosphere was discussed. These reactions have traditionally been studied experimentally in laboratory vessels called smog chambers. These chambers characteristically employ radiation sources that closely approximate the UV portion of the solar spectrum as observed at the earth's surface and clean, chemically inert interior surfaces. It is believed that the chemical processes that take place in smog chambers are similar to those that take place in the atmosphere.

The presence of surfaces in a smog chamber may, however, be a source of difficulty in interpreting chamber results because of possible surface-catalyzed reactions or absorption of species on the walls. In addition, most chamber experiments have been conducted by initially injecting fixed amounts of reactants rather than simulating the continuous time-varying injection and dilution of reactants that characterize the ambient situation. Nevertheless, the behavior of irradiated mixtures of oxides of nitrogen and hydrocarbons in smog chambers has served as the foundation for our understanding of atmospheric chemical mechanisms.

Considerable effort has been devoted to the development of chemical reaction mechanisms that are capable of describing the processes observed in smog chambers (Baldwin et al., 1977; Carter et al., 1978; Demerjian et al., 1974; Falls and Seinfeld, 1978; Whitten and Hogo, 1977). Smog chambers have been used extensively to determine how concentrations of NO_x and other photochemical products respond to changes in the initial composition of nitrogen oxides and organics. A previous Criteria Document (U.S. EPA, 1978) discusses smog chamber evidence concerning the relationship between ozone/oxidant and the photochemical precursors. This section focuses on how NO_2 concentrations respond to changes in the input levels of organics and nitrogen oxides.

Several researchers have used smog chambers to investigate the dependence of nitrogen dioxide concentrations on the levels of precursor inputs:

- The University of North Carolina (UNC) study, using an 11,000 cubic-foot (311 m^3) outdoor Teflon chamber, a simulated urban hydrocarbon mix, and twelve-hour irradiations (Jeffries et al., 1975)
- The Bureau of Mines study, using a 100 cubic-foot (2.8 m^3) aluminum-glass chamber, auto-exhaust hydrocarbons, and six-hour irradiations (Dimitriades, 1972,1977)
- The General Motors study, using a 300 cubic-foot (8.5 m^3) stainless steel-glass chamber, a simulated Los Angeles hydrocarbon mix, and six-hour irradiations (Huess, 1975)
- The University of California at Riverside study, using a 225 cubic-foot (6.4 m^3) glass chamber, a simulated Los Angeles hydrocarbon mix, and six-hour irradiations (Pitts et al., 1975)
- The Health, Education and Welfare (HEW) study, using a 335 cubic-foot (9.5 m^3) chamber, auto-exhaust hydrocarbons, and up to ten-hour irradiation time (Korth et al., 1964) and
- The HEW study, using a 335 cubic-foot (9.5 m^3) chamber, toluene and m-xylene, and 6-hour irradiations (Altshuller et al., 1970).

Trijonis (1978,1979) has recently reviewed the results of these studies, as summarized in Table 6-2. As indicated in Table 6-2, the various chamber studies basically agree concerning the dependence of maximum NO_2 and average NO_2 on NO_x input. With other factors held constant, maximum NO_2 and average NO_2 tend to be proportional to initial NO_x. The minor deviations away from proportionality that sometimes occur tend to be in the direction of a slightly less than proportional relationship, i.e., a 50 percent reduction in NO_x input sometimes produces slightly less than a 50 percent reduction in NO_2.

There is less agreement among the chamber studies concerning the dependence of NO_2 on initial hydrocarbon concentrations. With respect to maximum NO_2, the Bureau of Mines study indicates essentially no dependence on hydrocarbons. However, three other studies suggest that hydrocarbon reductions decrease maximum NO_2 concentrations. The UNC, General Motors, and UC Riverside studies indicate that 50 percent hydrocarbon control tends to decrease maximal NO_2 by 10-20 percent, 25 percent, and 10-15 percent, respectively.

With respect to average NO_2, the Bureau of Mines study indicates that hydrocarbon reductions would tend to increase NO_2 dosage. This result is consistent with the theoretical argument of Stephens (1973), who hypothesized that hydrocarbon reduction would increase average NO_2 because these reductions would delay and suppress the chemical reactions that consume NO_2 after it reaches a peak. However, the General Motors chamber study, the UC Riverside study, and the two HEW studies indicate that hydrocarbons produce no consistent effect on average NO_2 concentrations. The UNC experiments imply that a 50 percent reduction in hydrocarbons produces about a 20 percent decrease in average NO_2. There is some question about the UNC conclusion, however, because the UNC chamber runs were of a 10-hour duration and the NO_2 levels at the end of the experiments were greater when hydrocarbons were reduced. The extra NO_2 remaining after

TABLE 6-2. SUMMARY OF CONCLUSIONS FROM SMOG CHAMBER EXPERIMENTS

	MAXIMAL NO_2		AVERAGE NO_2	
CHAMBER STUDY	Dependence on NO_x	Dependence on HC	Dependence on NO_x	Dependence on HC
University of North Carolina (Jeffries et al., 1975)	Proportional or slightly less than proportional	50% HC reduction reduces maximal NO_2 by 10% to 20%	Proportional or slightly less than proportional	Uncertain, 50% HC reduction may decrease average NO_2 by 20% or may increase average NO_2
Bureau of Mines (Dimitriades, 1972,1977)	Proportional	No effect	Proportional	50% HC reduction increases average NO_2 by 10% to 30%
General Motors (Huess, 1975)	Slightly less than proportional	50% HC reduction reduces maximal NO_2 by 25%	Proportional to slightly less than proportional	No effect
UC Riverside (Pitts et al., 1975)	Proportional	50% HC control reduces maximal NO_2 by 10% to 15%	Proportional	No effect
HEW, Auto Exhaust (Korth et al., 1964)	----------	----------	Proportional	No consistent effect
HEW, Toluene (Altshuller et al., 1970)	----------	----------	Proportional	No effect

the 10-hour period could cause an increase in 24-hour average NO_2, even though average NO_2 was reduced during the first 10 hours.

Considering the results of all the chamber studies, Trijonis suggested a consensus based on existing chamber results which would appear to be as follows: fifty percent hydrocarbon reduction would have little effect on average NO_2 concentrations (a change of ± 10 percent) but would yield moderate decreases in maximal NO_2 (a reduction of about 10 to 20 percent). It should be noted that these conclusions are meant to apply to one basic type of ambient situation--the situation of well-mixed urban air.

Some additional support for these conclusions was provided recently by studies of actual ambient data on NO_x and hydrocarbon levels from a number of cities in the U.S. Using empirical modelling and historical trend analysis, Trijonis (1978,1979) concluded that the ambient data were generally consistent with the consensus of chamber results. The exact form of the NO_2/precursor relationship, however, was found to vary somewhat from one location to another, presumably depending on local hydrocarbon/NO_x ratios, on the details of the hydrocarbon mix, and on specific meteorological conditions.

Reference is made also to another body of data due to Pitts et al. (1977) (collected for a different purpose) which also contains potential information on the relationship between NO_x and its precursors. However, the data have not been analyzed to date for its pertinence to the NO_x precursor question.

6.1.3 NO_x Chemistry in Plumes

The atmospheric chemistry involving oxides of nitrogen in plumes from major fuel burning installations is essentially that described earlier. However, the relatively high concentrations of NO and NO_2 in such plumes compared with those in the ambient urban atmosphere leads to certain chemical phenomena particularly characteristic of plumes. For example, within or a few exit diameters downwind of a source such as the stack of a power plant or the exhaust system of a motor vehicle, the relatively high NO concentrations which may be present can produce NO_2 in significant amounts through reaction 6-1 given sufficient O_2. As another example, ambient ozone may be quickly scavenged in the plume by the large quantities of NO through reaction 6-4. Because the rate of the NO-O_3 reaction is fast relative to that of dilution of the plume, the rate of conversion of NO to NO_2 is controlled by the rate at which ambient O_3 is entrained into the plume by turbulent mixing (Hegg et al., 1976; Kewley, 1978; Shu et al., 1978; White, 1977). There is some nitric acid produced in power plant plumes during the daylight hours through the oxidation of nitric oxide (reaction 6-1) and the subsequent photodissociation of NO_2 (reaction 6-2), then followed by the combination of NO_2 with NO_3 and H_2O (reactions 6-10 and 6-8). The generation of nitrous acid is also probable since the stack gases will contain NO, NO_2, and H_2O (reaction 6-13). Since nitrous acid will photodissociate to give hydroxyl radicals (reaction 6-17), more nitric acid can be produced by reaction 6-18. Thus, although the free radical concentration is expected to be low in power plant plumes, some NO_x will be converted to nitric acid. In addition, after sufficiently long travel times during which ambient hydrocarbons have been mixed with the plume constituents, the usual free radical reactions described earlier occur, possibly leading to O_3 production.

There are several studies in which measurements have been made of the concentrations of pollutants in power plant plumes (Davis et al., 1974; Hegg et al., 1976; White et al., 1976). The most difficult current problem is predicting the rate at which NO is converted to NO_2 in such a plume.

6.1.4 Computer Simulation of Atmospheric Chemistry

A key problem underlying the development and evaluation of kinetic mechanisms for atmospheric chemistry is determining the sensitivity of the concentration predictions to those uncertain aspects of the reaction scheme. Such a determination can serve as a valuable guide for future experimental studies and for identifying those parameters that, when varied within accepted bounds, will be most influential on the predictions of the mechanism.

Although the qualitative aspects of the chemistry of the polluted troposphere appear to be reasonably well understood, there are many important details that still need to be investigated before a complete quantitative understanding of the photochemical smog system is possible. Several groups (Baldwin et al., 1977; Carter et al., 1978; Demerjian et al., 1974; Falls and Seinfeld, 1978; Whitten and Hogo, 1977) have formulated chemical reaction mechanisms for polluted tropospheric chemistry. Some of these are based on specific surrogate hydrocarbon chemistries; in others, attempts have been made to simulate the complex ambient atmospheric system by representing the general features of the hydrocarbon chemistry. All mechanisms contain aspects of uncertainty, whether in unknown rate constants, in the importance of competing reaction paths, or in the manner of representing the reaction of a generalized species. The measure of the accuracy of a mechanism is usually based on the extent of agreement between predicted concentration profiles and those generated experimentally in smog chambers.

With the recent elucidation of the chemistry of the reactions of OH and HO_2 with NO and NO_2, the inorganic portion of the photochemical smog mechanism is now, by and large, well understood. Uncertainties remaining include

- photolysis rates
- alkane-OH product distributions
- olefin-OH and olefin-O_3 product distributions
- aromatic chemistry
- alkoxyl radical reactions
- RO_x/NO_x reactions

A major uncertainty in the predictions lies in the specification of values of the photolysis rate constants. For analyzing smog chamber data, photolysis rate constants relative to the reported value for NO_2 are frequently used. Photolysis rate constants as a function of wavelength can be calculated from:

$$K_j = \sum_{0}^{\infty} \sigma_j(\lambda)\phi_j(\lambda)I(\lambda)\ d\lambda \qquad (6\text{-}45)$$

where

K_j = photolysis rate constant for species j
$\sigma_j(\lambda)$ = absorption cross section of species j
$\phi_j(\lambda)$ = quantum yield for the photolysis of species j
$I(\lambda)$ = actinic irradiance

Data applicable to some atmospheric systems have been compiled by Schere and Demerjian (1977). For species such as NO_2, HONO, and O_3, extensive experimental determinations of absorption cross sections are thought to be fairly reliable. However, since cross section and quantum yield data for formaldehyde, higher aldehydes, and alkyl nitrites are much less well characterized, many photolysis rate constants are subject to a large uncertainty. Of course, even if absorption cross sections and quantum yields could be determined accurately for all photosensitive species, uncertainties in atmospheric photolysis rate constants would still exist, as meteorological conditions, clouds, dust, and aerosols cause unknown variances in actinic irradiance.

Whereas rate constants in the inorganic portion of the mechanism are known fairly well, many more uncertainties, both in reaction rate constants and products, are associated with the organic reaction steps. Still to be determined are product distributions and reaction rate constants for the initial steps of the reactions of OH and hydrocarbon species, the largest uncertainties lying in the routes of the various radical species produced. For example, although rate constants for alkane-OH reactions are well established, the ratio of internal to external abstraction for all alkanes is not known. Addition to O_2 to form peroxyalkyl (RO_2) radicals can be considered as the sole fate of the alkyl radicals first produced in alkane-OH reactions, but after the formation of alkoxyl radicals through the conversion of NO to NO_2, the reaction mechanism becomes uncertain. Alkoxyl radicals can decompose, react with O_2, isomerize, or react with NO or NO_2, with the importance and rate of each reaction path depending on the nature of the alkoxyl group. Even for the most studied of the alkane-OH reactions, the relative rates between decomposition, isomerization, and reaction with O_2, NO, and NO_2 for alkoxyl radicals have not been measured, but must be estimated (Baldwin et al., 1977).

Less well understood than alkane reaction mechanisms are olefin oxidation processes, primarily by OH. Olefin-OH reactions may proceed by addition or abstraction. For smaller olefins, the addition path predominates. However, the abstraction fraction increases with the size of the olefin. Along the addition path for terminally bonded olefins, there is uncertainty as to the ratio of internal to external addition. Similar to alkyl radicals, the hydroxy-alkyl radicals formed in the initial OH addition to olefins are thought to immediately add O_2 to form hydroxy-peroxyalkyl radicals and thereafter react with NO to give NO_2 and hydroxy-alkoxyl species. The fate of the hydroxy-alkoxyl radicals is subject to speculation, although the analogous alkoxyl reaction paths of decomposition, isomerization, and reaction with NO, NO_2 and O_2 are most likely possibilities.

The inherent uncertainty of the decomposition, reaction with O_2, and isomerization of the alkoxyl and hydroxy-alkoxyl radicals class can be represented by the generalized reaction step:

$$RO \rightarrow \alpha HO_2 + (1-\alpha)RO_2 + \beta HCHO + \gamma RCHO \qquad (6\text{-}46)$$

From the earlier discussions of alkoxyl radical behavior, RO always gives rise to either HO_2 or RO_2 in any of the decomposition, isomerization, or O_2 reaction pathways. Hence, the stoichiometric coefficients representing the fraction of HO_2 and RO_2 found in the lumped RO reaction should sum to one. Since the RO lumped species represents a large class of different-sized radicals and because splits between reaction paths for even specific radicals are unknown, α can have a value in the range 0 to 1. Many RO reaction routes produce aldehydes. Thus, $0 \leq \beta \leq 1$ and $0 \leq \gamma \leq 1$. Since the composition of the RO radical pool is continually changing during the course of a photooxidation, the actual values of α, β, and γ are functions of time. Thus, the selection of constant values of these coefficients introduces uncertainty.

A comprehensive sensitivity/uncertainty analysis of photochemical smog mechanisms has been carried out by Falls et al. (1979). In this study the effects of rate constant and mechanistic unc tainties on predicted concentrations are illustrated.

6.2 NITRITE AND NITRATE FORMATION

The oxides of nitrogen are converted eventually to nitrites and nitrates by the reactions given in Section 6.1. In particular, the following gaseous nitrites and nitrates have been identified:

HONO	nitrous acid
$HONO_2$	nitric acid
HO_2NO_2	peroxynitric acid
RONO	alkyl nitrite
$RONO_2$	alkyl nitrate
$R\overset{O}{\overset{\parallel}{C}}OONO_2$	peroxyacylnitrate (PAN)
RO_2NO_2	peroxyalkyl nitrate

In addition to these gaseous species, particulate nitrites and nitrates may be formed. The object of this section is to present estimates of the importance of the various nitrites and nitrates. In most cases, estimates are necessary because ambient measurements of the concentration level of all but a very few of the species are lacking.

Typical ambient concentration levels of the gaseous nitrogen-containing species listed above can be estimated from simulations of smog chamber experiments using chemical mechanisms representing the hydrocarbon-NO_x chemistry. Table 6-3 lists calculated concentrations of HONO, $HONO_2$, HO_2NO_2, RONO, $RONO_2$, $RC(O)OONO_2$, and RO_2NO_2 for smog chamber experiment EC-237 carried out at the Statewide Air Pollution Research Center of the University of California, Riverside, using the chemical mechanism of Falls and Seinfeld (1978). The conditions of the experiment are given in the footnote of Table 6-3. The simulated and predicted concentrations of the major measured species, such as NO, NO_2 O_3, PAN, and hydrocarbons, agreed well.

TABLE 6-3. PREDICTED NITRITE AND NITRATE CONCENTRATIONS IN SIMULATION OF EXPERIMENT EC-237 OF THE STATEWIDE AIR POLLUTION RESEARCH CENTER OF UNIVERSITY OF CALIFORNIA, RIVERSIDE, USING THE CHEMICAL MECHANISM OF FALLS AND SEINFELD (1978)

	Concentration, ppm		
	60 min.	180 min.	300 min.
HONO	0.0061	0.00040	0.00036
$HONO_2$	0.067	0.22	0.29
HO_2NO_2	0.00083	0.0019	0.0025
RONO	0.0030	0.00054	0.000080
$RONO_2$	0.0041	0.0070	0.0072
$\mathrm{R\overset{O}{\overset{\|}{C}}OONO_2}$	0.025	0.089	0.13
RO_2NO_2	0.034	0.075	0.098

Conditions of the experiment: $T = 303^oK$, $k_2 = 0.3\ min^{-1}$, $[NO_2]_o = 0.106$, $[NO]_o = 0.377$, $[H_2O] = 2.4 \times 10^4$, $[CO]_o = 0.96$, $[Aldehydes]_o = 0.0012$, $[Alkanes]_o = 1.488$, $[Non\text{-}ethylene\ Olefins]_o = 0.15$, $[C_2H_4]_o = 0.875$, $[Aromatics]_o = 0.177$, $[HONO]_o$ (assumed) $= 0.1$ (All concentrations in ppm). Dilution rate $= 2.93 \times 10^{-4}\ min^{-1}$

The concentrations of HONO, HO_2NO_2, and RONO are predicted to be small relative to those of NO and NO_2. Each of these species has decomposition reactions,

$$HONO + h\nu \rightarrow OH\cdot + NO \qquad (6\text{-}17)$$

$$HO_2NO_2 \rightarrow HO_2\cdot + NO_2 \qquad (6\text{-}36)$$

$$RONO + h\nu \rightarrow RO\cdot + NO \qquad (6\text{-}47)$$

that, at the temperatures and solar intensities prevalent in the experiment and in the summer atmosphere, are fast enough to insure that the concentrations of each of the three species are low. At lower solar intensities than those in the experiment, HONO and RONO can be expected to reach higher concentrations, and at lower temperatures, such as those in the stratosphere, HO_2NO_2 may accumulate.

Under daytime conditions the reactions that govern the concentration of HONO are 6-17 and 6-19. At night, however, the only apparent destruction route for HONO is reaction 6-14. Depending on the relative importance of reactions 6-19, 6-13, and 6-14, HONO may reach substantial concentrations under nighttime conditions. A lower limit on the nighttime concentration of HONO can be estimated from the equilibrium HONO concentration based on reactions 6-13 and 6-14.

$$[HONO] = \frac{K_{13}[NO][NO_2][H_2O]}{k_{14}}^{1/2} \qquad (6\text{-}48)$$

At $[NO] = [NO_2] = 0.1$ ppm, $[H_2O] = 2.4 \times 10^4$ ppm (50 percent relative humidity), the equilibrium HONO concentration calculated from equation 6-48 is 1.9×10^{-2} ppm.

Like HONO, HO_2NO_2 and RONO, PAN undergoes both formation and decomposition steps (reactions 6-42a,b). Unlike these former species, however, the balance between the formation and decomposition reactions is such that PAN may achieve appreciable concentration levels relative to those of NO and NO_2. Because the decomposition reaction for PAN is strongly temperature dependent, the steady state PAN concentration is highly dependent on the temperature. As temperature increases the role of PAN as an NO_2 sink decreases markedly; at low temperatures, on the other hand, steady state PAN concentrations can reach rather substantial levels.

Little is known about the existence and importance of peroxynitrates other than HO_2NO_2 and PAN. It was presumed in the mechanism on which the results of Table 6-3 are based that RO_2NO_2 thermally decomposes at a rate between those for HO_2NO_2 and PAN. Assessment of the importance of RO_2NO_2 as a sink for NO_x will depend on measurement of the rates of reactions 6-40 and 6-41.

In contrast to the other species of Table 6-3, nitric acid and alkyl nitrates apparently do not undergo appreciable decomposition reactions. Thus, these two species potentially serve as important atmospheric sinks for NO_2. Both nitric acid and alkyl nitrates may remain in the gas phase or react with other atmospheric constituents, such as ammonia, to produce low vapor pressure species that have a tendency to condense on existing particles or homogeneously nucleate to form particles.

Figure 6-1 depicts the potential paths by which particulate nitrate species may be formed from NO and NO_2. Path 1 involves the formation of gaseous nitric acid by reactions 6-8 and 6-18. Nitric acid concentrations resulting from these two reactions for the simulated smog chamber experiment have been given in Table 6-3. Comparisons of the individual rates of reactions 6-8 and 6-18 indicate that reaction 6-18 is the predominant route for gas-phase nitric acid formation under typical daytime conditions. Nitric acid vapor, once formed, may then react with NH_3, a ubiquitous atmospheric constituent with both natural and anthropogenic sources, to produce ammonium nitrate, NH_4NO_3 (path 2), which at standard temperature and pressure, exists as a solid. Alternatively, the nitric acid vapor may be absorbed directly into a particle (path 3), although thermodynamic and kinetic considerations favor reaction with NH_3 to form NH_4NO_3 as the path of conversion of gaseous nitric acid to nitrate in particulate form (Bradner et al., 1962; Morris and Niki, 1971; Stelson et al.). Path 4 involves the direct absorption of NO and NO_2 into an atmospheric particle, a route that is likely for certain aqueous particles, particularly when accompanied by the absorption of ammonia (path 5) (Orel and Seinfeld, 1977). Path 6 depicts the formation of organic nitrates through reactions

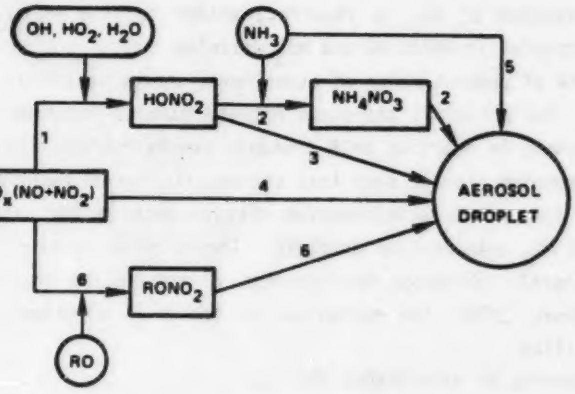

OH, HO₂, H₂O
NH₃
HONO₂
NH₄NO₃
NOₓ (NO+NO₂)
RONO₂
RO
AEROSOL DROPLET
1
2
2
3
4
5
6
6

Figure 6-1. Paths of nitrate formation in the atmosphere (Orel and Seinfeld, 1977).

such as 6-38a, followed by absorption of these nitrates into particles. At present little is known about the existence or importance of mechanisms such as that depicted by path 6.

There have been a number of measurements of nitric acid and particulate nitrate concentrations in ambient air, and several of these are summarized in Chapter 8. Many of the measurements have identified the particulate nitrate as NH_4NO_3, suggesting that the aerosol may consist of solid NH_4NO_3 or NH_4^+ and NO_3^- in solution in approximate stoichiometric balance. It is difficult to estimate the relative importance of the paths in Figure 6-1 for several reasons. First, the rate of reaction of nitric acid and ammonia is not well known, although the forward reaction is probably rapid and, in fact, can be presumed to be in equilibrium with the dissociation of solid ammonium nitrate (Bradner et al., 1962; Morris and Niki, 1971; Stelson et al.).

$$NH_{3(g)} + HONO_{2(g)} \rightleftharpoons NH_4NO_{3(s)} \qquad (6\text{-}49)$$

Second, the rate of absorption of NO and NO_2 into existing particles depends on the composition and size of each particle and cannot generally be predicted a priori. In either case it is apparent that the presence of NH_3 is required, either to form NH_4NO_3 or to neutralize the acidity of a liquid droplet in which NO and NO_2 dissolve.

The current state of understanding of atmospheric inorganic nitrate formation can be summarized as follows. The principal gas-phase nitrate forming reaction is reaction 6-18. The nitric acid vapor formed in reaction 6-18 probably reacts rapidly with ammonia to form small particles of solid ammonium nitrate such that the equilibrium of reaction 6-49 is established. In competition with the nitric acid/ammonium nitrate path is the path consisting of direct absorption of NO and NO_2 into aqueous droplets. The relative rates of these two paths cannot be determined in general. Although measurements of particulate organic nitrate levels have been reported (Grosjean, 1979), the mechanisms of formation of organic aerosol nitrates have not been fully identified.

6.3 TRANSPORT AND REMOVAL OF NITROGENOUS SPECIES

The general behavior of nitrogenous species in the atmosphere can be described as follows. Nitric oxide emissions are converted partially to nitrogen dioxide within the urban atmosphere as a result of gas-phase reactions. Simultaneously, NO_2 is converted to nitric acid vapor and NO and NO_2 may also be absorbed into existing particles. The mixture of gases and particles is transported downwind of the source region, accompanied by continuous conversion of more of the NO_x gases to particulate nitrates. Also occurring simultaneously is surface absorption of NO and NO_2 as well as of particles containing nitrate. Eventually, rainout and washout serve to remove more of the remaining gases and particles.

The object of studying the transport and removal processes of nitrogenous species is to develop the capability to predict the atmospheric residence time of nitrogenous species as they are transported downwind from a source-rich area. Several recent studies have been reported in which measurements (usually airborne) have been carried out downwind of large urban complexes in order to obtain material balances on gaseous and particulate pollutants (Breeding et al., 1976; Stampfer and Anderson, 1975). A goal of these studies is to determine the relative roles of transport, removal and conversion of gaseous to particulate pollutants on the overall pollutant material balance downwind of a major urban source. On the basis of the previous discussion in this section it is possible to make rough estimates of the relative roles of these processes in determining the ultimate fate of nitrogenous species.

In the quantitative analysis of urban plume data, it is necessary to have a mathematical model capable of describing the behavior of both gaseous and particulate pollutants and their interrelations. Such a model would, in principle, include both gaseous and particulate phases with detailed treatments of gas-phase and particulate-phase chemistry, as well as size distributions of the particles.

There currently exist a number of mathematical models capable of relating emissions of primary gaseous species to airborne concentration levels of both primary and secondary gaseous pollutants. The models include details of atmospheric chemistry and meteorology and have been exercised in a variety of situations. Models that relate emissions of primary and secondary gaseous and particulate pollutants, including description of particle size distribution and chemical composition, are currently under development and not yet available for general use. In view of this, a "first-order" model that contains all the major mechanisms influencing the airborne concentrations of gaseous and particulate pollutants can be formulated, one that does not include details of atmospheric chemistry and particle size distribution, but treats the competing processes of advection, turbulent diffusion, conversion of gaseous species to particulate material, settling, deposition, washout and rainout. Such a "first-order" model is in essence a material balance, designed to provide estimates of the fraction of pollutants that still remains airborne at a certain distance downwind of the source and the fraction that has been removed by deposition and gas-to-particle conversion. A model of this type has been developed by Peterson and Seinfeld (1977) and applied to the prediction of airborne concentrations of gaseous and particulate pollutants in the case in which gases are converted to secondary particulate matter. Although the model of Peterson and Seinfeld is a quantitative framework within which to evaluate each of these effects, at this time only qualitative estimates for nitrogen-containing species are possible because of lack of knowledge of the relevant rates and coefficients for such a model. The execution of experimental measurements in urban plumes and correlation with physical and chemical rate data to predict fractions of nitrogenous species that are removed by various paths has yet to be performed. See Section 6.3.3 for further discussion.

In this section the general nature of the transport and removal of nitrogeneous species is briefly discussed.

6.3.1 Transport and Diffusion

Some atmospheric processes play an important role in the dispersion of air pollutants on large spatial scales, and others are important on small spatial scales. The interactions among these processes, and their overlapping influences on eventual pollutant distributions, are very complex. A classical example, shown in Figure 6-2, is the effect of atmospheric turbulence of different scales on pollutant transport and dispersion.

The spatial and temporal scales of interest to the long-range transport of nitrogenous and other pollutant species are on the order of several hundred kilometers and several days. As shown in Figure 6-2, the atmospheric motions important on these scales range from mesoscale convection to synoptic-scale cyclonic waves.

Changes of wind speed and direction in the lowest layer of the atmosphere are the result of many competing physical processes. The interaction between the synoptic-scale air motion and the surface boundary layer usually produces complex flow patterns. These patterns change hourly, daily and seasonally. They also vary spatially, especially where terrain is nonuniform and the heating or cooling of the surface is inhomogeneous. Vertical air motions result from divergence in synoptic and mesoscale wind flow. They are also produced by viscous and frictional forces in the boundary layer and can be particularly large and highly variable in regions of complex terrain. Although vertical velocities generated by these processes have a magnitude of only 1-10 centimeters per second, which is 1-2 orders of magnitude less than generally observed horizontal wind speeds near the surface, they have significant effects on the net transport and dispersion of air pollutants. Accurate estimates of the vertical components of the wind vectors are extremely difficult to obtain on a routine basis, particularly in the first few hundred meters of the surface. Further, they vary drastically with horizontal wind speed and the radiation balance of the surface. While vertical diffusion is dominant generally in the first 10 to 30 km away from the source, lateral (or horizontal) diffusion becomes important in pollutant transport over large distances. Vertical diffusion in the troposphere is limited to about 10 km in mid-latitudes while the lateral transport eventually varies through 360 degrees.

6.3.2 Removal Processes

The atmospheric residence times of nitrogenous species (lifetimes) are of the order of days to weeks, while their inter-specie lifetimes (chemical transformation) might only be as short as fractions of a second.

Atmospheric residence times are governed by the efficiency with which species are removed from the atmosphere. The removal (or cleansing) efficiency depends considerably on the specific physical and chemical nature of the species. For example, NO, NO_2, and HNO_3 are removed within clouds and/or by rain with different efficiencies due to the different solubility and vapor pressure characteristics of the three gases.

There are two types of removal processes that occur in the atmosphere:

- dry deposition
- precipitations scavenging (wet deposition)

Mathematical models capable of describing the behavior of both gaseous and particulate pollutants must include removal terms, particularly for the important nitrogenous species, such as nitric acid and nitrate aerosol.

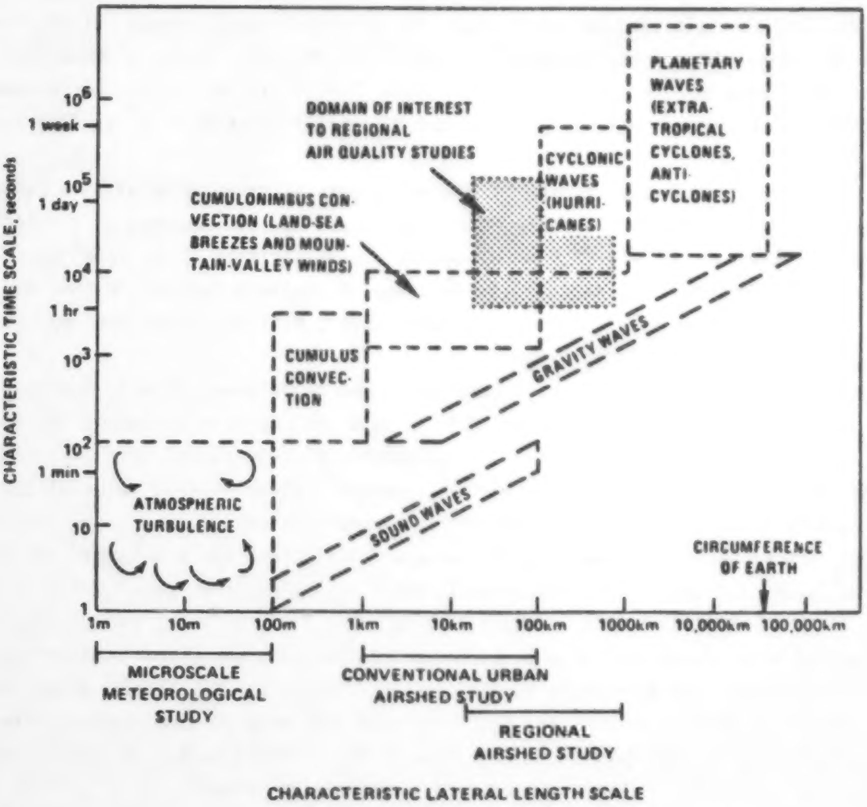

CHARACTERISTIC TIME SCALE, seconds
10⁶
1 week
10⁵
1 day
10⁴
1 hr
10³
10²
1 min
10
1
1m
10m
100m
1km
10km
100km
1000km
10,000km
100,000km
PLANETARY WAVES (EXTRA-TROPICAL CYCLONES, ANTI-CYCLONES)
DOMAIN OF INTEREST TO REGIONAL AIR QUALITY STUDIES
CYCLONIC WAVES (HURRI-CANES)
CUMULONIMBUS CONVECTION (LAND-SEA BREEZES AND MOUNTAIN-VALLEY WINDS)
CUMULUS CONVECTION
GRAVITY WAVES
SOUND WAVES
ATMOSPHERIC TURBULENCE
CIRCUMFERENCE OF EARTH
MICROSCALE METEOROLOGICAL STUDY
CONVENTIONAL URBAN AIRSHED STUDY
REGIONAL AIRSHED STUDY
CHARACTERISTIC LATERAL LENGTH SCALE

Figure 6-2. Schematic illustration of scales of motion in the atmosphere.

6.3.2.1 *Dry Deposition of Gases*--Gaseous nitrogeneous species are removed from the atmosphere by surface absorption, so-called deposition. The rate at which an airborne species of concentration c_i (micrograms per cubic meter) is removed across a horizontal surface of unit area at an elevation z_s is often expressed as $c_i v_i$ where v_i is the so-called "deposition velocity," which depends on the value of z_s.

Vegetation has been shown capable of removing significant amounts of NO_2 and NO from the atmosphere. Tingey (1968) showed that alfalfa and oats absorbed NO_2 from the air in excess of 100×10^{-12} moles per square meter per second when exposed to an atmosphere containing 460 $\mu g/m^3$ NO_2 (or 1×10^{-5} moles per cubic meter). More recent work by Rogers et al. (1977), using a continuous reactor technique, indicated that the NO_2 uptake in both corn (*Zea mays* l.) and soybean (*Glycine* max. L.) could be well represented by a chemical kinetic model having two reactants, the pollutant and the leaf surface. The second order rate constant for NO_2 uptake was independent of NO_2 concentrations and leaf surface area, but directly dependent upon inverse total diffusion resistance. Tingey (1968) extrapolated his data to estimate the removal of NO_2 from the Salt Lake Valley in Utah. On an annual basis, removal of NO_2 from the valley with an ambient NO_2 concentration of 9.6 $\mu g/m^3$ (0.005 ppm), which is about 3-4 times greater than background NO_2 levels, would total about 3×10^6 kg NO_2. This may be compared with an estimated total global anthropogenic annual NO_2 emissions of 53×10^9 kg (Robinson and Robbins, 1970).

Hill (1971) found in his experiments on the uptake rate of gases by an alfalfa canopy that NO was absorbed with a deposition velocity of 0.1 cm/sec and NO_2 was absorbed at a velocity of 2 cm/sec when present in the air of the chamber at a concentration of 96 $\mu g/m^3$ (0.05 ppm). Using ambient NO_2 concentrations found in those areas of Southern California from August to October of 1968, and assuming a continuous alfalfa cover, Hill estimated that NO_2 could be removed at a rate of 0.1 gram/m^2/day.

Nitrogen oxides (especially N_2O) have long been known to be produced by biological action in soils. Recently, however, Abeles et al. (1971) found that soils could absorb NO_2 from the atmosphere as well. They found that when air containing NO_2 was passed over soil in a test chamber, the concentration of NO_2 in the air was reduced from an initial value of 190×10^3 $\mu g/m^3$ (100 ppm) to 5.7×10^3 $\mu g/m^3$ (3.0 ppm) over a 24-hour period. When soil was autoclaved, the total NO_2 present over the same time period was reduced from 186×10^3 $\mu g/m^3$ (97 ppm) to only 25×10^3 $\mu g/m^3$ (13 ppm). This result would point to a biological sink for NO_2 in soils. Ghiorse and Alexander (1976), however, report finding essentially no difference in NO_2 removal by soil from air in a closed system when their soil (Lima loam) was either nonsterile, autoclaved or γ-irradiated. The authors point out that autoclaving may drastically alter the physical and chemical properties of soil and thus introduce artifacts in such studies. They conclude that the role of microorganisms in the fate of NO_2 in soils is not so much in sorption but in conversion of nitrite (resulting from such sorption) into nitrate.

Nitric oxide may be absorbed by soils, but is then oxidized almost immediately to NO_2. Mortland (1965) has noted that transition metal ions in the soil promote NO absorption. If the soil is saturated with alkaline earth cations though, absorption of NO is halted. Sundareson et al. (1967) found that alkaline-earth zeolites readily absorb NO and release it as NO_x and HNO_3 when heated. To date, the role of organic matter in the absorption of nitrogen oxides by soil is unknown.

6.3.2.2 Dry Deposition of Particles--The deposition of particles can occur through sedimentation, Brownian diffusion, or impaction. Impaction occurs when, because of its inertia, a particle is unable to follow the streamlines of air around an obstacle and is intercepted by the object. The removal of particles through impaction on an object can be defined in terms of a pseudo-deposition velocity, v_g. The loss of particles per unit surface area of the object per unit time can then be expressed as:

$$L_D^A = -v_g N, \qquad (6\text{-}48)$$

where N is the number density of particles in the size range corresponding to the deposition velocity, v_g.

The transfer of aerosol particles from the turbulent atmosphere to an underlying boundary depends upon the flow near the surface, as well as upon the nature of the surface itself. Particles are transferred through a turbulent boundary layer, the transport properties of which depend on the eddy motion of the turbulence. Near the surface, the particles move through a laminar sublayer, where the thermal motion of the particles becomes important.

Particle removal from the atmosphere by deposition strongly depends on the properties of the surface on which material deposits, the surface roughness, and the wind speed. For the purposes of an order-of-magnitude estimate of deposition rates, one can use the results of Chamberlain (1967) to estimate v_g.

6.3.2.3 Wet Deposition--Precipitation can remove gases and particles by two methods, rainout and washout. Rainout involves the various processes taking place within a cloud that lead to the formation of raindrops. Washout refers to the removal of aerosols below the cloud by falling raindrops. Wet deposition removal effectiveness probably varies with the form of the precipitation, e.g., liquid *versus* solid forms. Rainout and washout, together with dry deposition, are the major sinks for atmospheric nitrogen-containing species.

Particles are removed by rainout through their serving as condensation nuclei for cloud formation. The extent of absorption of gases by cloud droplets depends on the chemical compositions of both the gases and the droplets. Whereas the removal of SO_2 by cloud droplets has received considerable attention, the processes taking place during the absorption of NO and NO_2 by water droplets have not yet been thoroughly studied. In a study of aerosol nitrate formation routes, Orel and Seinfeld (1977) elucidated many of the chemical processes that occur when NO and NO_2 are absorbed in water droplets. The most important factor in the overall efficiency of rainout in removing oxides of nitrogen from the atmosphere is the frequency of rains. Because of the difficulty in describing the detailed processes occurring in rainout, it is generally assumed that removal of pollutants by rainout can be described adequately by a characteristic mean residence time, and that the amount of pollutant removed by rainout at any one place is proportional to the ambient concentration of that pollutant.

The capture of gases and particles by falling raindrops is called washout. Typically, the duration of washout is relatively short compared with that of rainout. However, pollutant concentrations at the cloud level are frequently much lower than those near the ground where washout occurs. Thus, rainout and washout can be of similar importance. The uptake of NO_x by rain depends on physical parameters such as rainfall intensities and raindrop size distributions, and on the chemical composition of the raindrops. Models of washout generally reduce to two limiting cases, mass-transfer-limited and chemical-reaction limited. In the former, the rate controlling step for absorption is the diffusion of the gases to the falling drop; in the latter, chemical equilibrium in the drop controls the quantity absorbed. The study of Dana et al. (1973) suggests that for SO_2 under typical atmospheric conditions washout is often mass-transfer-limited. Similar studies for washout of NO_x have yet to be performed.

6.3.3 Source-Receptor Relationships

The previous sections of this chapter have addressed the scientific basis for our current understanding of the atmospheric chemical processes describing the transformation of NO, which is the dominant NO_x compound emitted by most sources, into the more toxic NO_2, and its subsequent further transformation, transport and removal. This section addresses the question of predicting the NO_2 concentration experienced by a receptor, such as a human being, due to given emissions of NO_x (see Chapter 5). Methodology for estimating human exposure using ambient concentration data derived from fixed monitoring sites is discussed in Chapter 8.

Until very recently, the problem of predicting NO_2 concentrations has received little attention in the literature. However, relationships among ambient nitrogen oxides, hydrocarbons (HC) and ozone/oxidant (O_x) have been considered in connection with the question of predicting ambient ozone concentrations (U.S. EPA, 1978). Some of the methodology applicable to the ozone problem has potential usefulness in the case of NO_x source-receptor relationships. In this regard two very recent reviews are available (Cole and Summerhays, 1979; Anderson et al., 1978). In addition, a critical review of atmospheric modeling has also been published (Turner, 1979).

Relationships between emissions and O_x/NO_x-related air quality have been pursued following three distinct approaches that differ mainly in degree of empiricism. In order of decreasing empiricism, these approaches are as follows:

(1) Empirical Approach. This approach entails statistically or nonstatistically associating ambient air quality data either with ambient concentrations of precursors or with precursor emission rates. These associations are clearly not cause-effect in nature, and their intended use is not to predict absolute air quality; rather, it is to estimate changes in air quality resulting from changes in emission rates.

(2) Mechanistic Models of $O_x/HC/NO_x$. This approach entails deriving cause-effect relationships between oxidant and precursors through laboratory testing and chemical mechanistic simulations. As in the preceding case, this approach is intended to predict only changes in air quality resulting from changes in emission rates.

(3) Air Quality Simulation Model (AQSM) Approach. This approach entails deriving the requisite air quality-emission relationships through mathematical representation of the transport, dispersion, transformation, and deposition processes. Its intended use is to predict absolute levels of air quality from given emission rates and meteorological data.

The air quality-emissions relationships or "models" developed to date through all these approaches are applicable only to the urban problem, or to situations in which the geographical dimension of the source-receptor relationship is comparable to that of the urban area. Techniques for estimating short-term NO_2 concentrations arising from point sources alone have been discussed by Cole and Summerhays (1979).

Since the first two approaches predict only changes in air quality resulting from changes in emission rates rather than absolute levels of air quality, their utility lies mostly in the technical area of air quality management. The third approach is most pertinent to the discussion in this section.

If predictions from AQSM models could be made reliably and with a reasonable effort, it would be possible, e.g., to:

- Determine the impact of different source types and/or individual sources upon absolute air quality.
- Augment ambient monitoring data with calculated data at points intermediate to the limited number of monitoring sites practically available. Such new data would be useful in a number of applications including the location of "hot spots" and the estimation of human exposure to NO_2 concentrations.

Assessment of the specific impacts on air quality of the various source types in the NO_2 problem requires consideration of such parameters as local pollutant sources and/or concentrations, meteorology, and topography, all of which vary from area to area. Development and validation of AQSM models applicable to the NO_2 problem, currently underway at the USEPA and elsewhere, have reached a stage where it is now becoming possible to evaluate their usefulness and accuracy. At the present time, however, the literature contains little documentation of specific applications of AQSMs. For this reason, a meaningful discussion of source impacts on air quality could not be included in this document.

In general, the utility of model types for the above purposes depends not only on the completeness with which they describe the chemical and physical processes characterizing the NO_2 problem, but also on the type of situation to be modelled. For example, prediction of NO_2 concentrations very close to a highway in the absence of all other NO_x sources except lines of mobile vehicles might, for practical purposes, be made using a model which did not take into account the free radical chemistry described previously since these reactions would probably not have time to occur. On the other hand, prediction of NO_2 concentrations on an urban scale resulting from a combination of point and area sources would probably require consideration of the complex chemistry reviewed in this chapter.

In summary, it may be stated that the prospects for estimating NO_2 concentrations at local receptor points are quite promising but it is not possible to estimate reliably their accuracy or usefulness based on currently reported applications.

6.4 MECHANISMS OF ATMOSPHERIC NITROSAMINE FORMATION

This section is limited to a discussion of specific reactions possibly leading to the formation of nitrosamines and related compounds in the atmosphere.

Three mechanisms will be discussed in this section:

- Non-photochemical reactions of gaseous amines with oxides of nitrogen and nitrous acid
- Photochemical reactions of amines with oxides of nitrogen in the gas phase
- Heterogenous nitrosamine formation processes in atmospheric aerosols

The first two processes have been the object of recent experimental studies, including simulation experiments in environmental chambers, and will be examined in some detail. The third process involving aerosol particles is purely speculative at this time and will be briefly discussed in terms of the corresponding evidence in the bulk (liquid) phase.

6.4.1 Non-Photochemical Reaction of Gaseous Amines with Oxides of Nitrogen and Nitrous Acid

Bretschneider and Matz (1973,1976) reported the fast formation of diethylnitrosamine (DENA) when reacting 50-100 ppm of diethylamine and nitrogen dioxide. DENA formed within seconds and was reportedly stable for weeks in the dark in the glass reaction vessel. Dimethylnitrosamine was formed in the same way from dimethylamine and NO_2. The authors also report that nitrosamine formation can be catalyzed by SO_2.

A fast reaction between diethylamine and NO_2 was also reported by Gehlert and Rolle (1977), who achieved in a few minutes a 90 percent conversion to diethylnitrosamine at 25°C. They proposed the following rate equation:

$$-d(NO_2)/dt = k\ (\text{dimethylamine})\ (NO_2)^2 \qquad (6\text{-}51)$$

with a rate constant $k_{51} = 6.5 \times 10^8\ \ell^2\ \text{Mol}^{-2}\ s^{-1}$. Initial reactant concentrations ranged from 4×10^{-6} to 6×10^{-5} Mol ℓ^{-1}. The other major product of the reaction was the amine nitrate (aerosol), corresponding to the overall equation:

$$2\ NO_2 + 2(C_2H_5)_2NH \rightarrow (C_2H_5)_2NNO + (C_2H_5)_2NH_2NO_3 \qquad (6\text{-}52)$$

Neurath and co-workers (1965,1976) investigated the effect of adding several amines on the thermal oxidation of nitric oxide to nitrogen dioxide in the presence of 10 percent oxygen. Addition of secondary amines (dimethyl, diethyl, methyl-n-butyl, and pyrrolidine) doubled the rate of NO oxidation, presumably due to nitrosamine formation:

$$NO + NO_2 \rightarrow N_2O_3 \qquad (6\text{-}53)$$

$$N_2O_3 + 2\ R_2NH \rightarrow 2\ R_2NNO + H_2O \qquad (6\text{-}54)$$

where R = alkyl group. Addition of tertiary amines (trimethyl, diethyl-methyl, and N-methyl pyrrolidine) also increased the NO oxidation rate, thus indicating that tertiary amine also reacted with the oxides of nitrogen under these conditions. The reactions of dimethylamine and diethylamine with NO in the presence of 10 percent oxygen were also followed by directly measuring the (decreasing) amine concentration. No reaction was observed between diethylamine and NO in nitrogen.

Dushumin and Sopach (1976) also report a rapid reaction between dimethylamine, N_2O_4 (in equilibrium with NO_2) and ozone to form dimethylnitrosamine with a 50 percent conversion achieved in less than 10 minutes. Since other reaction products included formaldehyde (HCHO) and dimethylnitramine $[(CH_3)_2NNO_2]$, it is suspected that photochemical reactions took place as well, as is discussed in the next sub-section (6.4.2). Field measurements were performed near a chemical complex, and dimethylnitrosamine was found in the frost near the complex as well as in the air up to 30 kilometers from the complex.

In complete contrast with the above studies, low yields of nitrosamines were obtained by Hanst et al. (1977), Grosjean et al. (1977), and Pitts et al. (1978) in experiments involving dark reactions of ppm levels of alkylamines and nitrogen oxides in humid air.

Hanst, Spence and Miller (1977), using long path infrared spectroscopy, followed the reaction of 1 ppm dimethylamine with 0.5 ppm HONO (in equilibrium with 2 ppm NO, 2 ppm NO_2 and 13,000 ppm water vapor) in air in a 9 x 0.3 m diameter cylindrical glass cell. Dimethylnitrosamine was formed in yields of 10 to 30 percent, and the rate of amine disappearance was ~4 percent min^{-1}.

Grosjean et al. (1977) and Pitts et al. (1978) also report low yields of nitrosamine in the dark reaction of ~0.5 ppm amine with 0.8 ppm NO and 0.16 ppm NO_2 in air at 30 percent relative humidity in 50 m^3 Teflon chambers. Nitrosamine yields were 2.8 percent from diethylamine and ~1 percent from dimethylamine. The tertiary amine triethylamine also yielded diethylnitrosamine (0.8 percent yield), while trimethylamine yielded traces of dimethylnitrosamine. For all four amines the nitrosamines were the only gas phase products found after 2 hours of reaction in the dark. Light-scattering aerosols were also formed.

Assuming a bimolecular reaction between the amine and nitrous acid, and using the nitrous acid equilibrium constant of Chan et al. (1967), Hanst et al. (1977) estimated an amine disappearance rate of 0.8 ppm min^{-1}. This would constitute an upper limit for nitrosamine formation since (a) the nitrosamine yield is not necessarily 100 percent of the reacted amine, (b) some of the amine may be lost on the walls of the reaction vessel rather than by chemical reaction, and (c) nitrous acid formation may be controlled by heterogeneous rather than homogeneous processes.

With respect to the latter, Cox and Derwent (1976/77) reported decomposition of 150 ppm HONO at a rate of 10 to 15 ppm hr^{-1}, i.e., ~ 200 times slower than the rate predicted using the data of Chan, et al. More recently, Kaiser and Wu (1977) reinvestigated the kinetics of formation and decomposition of nitrous acid:

$$NO + NO_2 + H_2O \rightarrow 2\ HONO \qquad (6\text{-}55)$$

$$2\ HONO \rightarrow NO + NO_2 + H_2O \qquad (6\text{-}56)$$

They found the reactions to be heterogeneous under all surface conditions tested. They also estimated upper limits for the homogeneous rate constants:

$$k_1 \leq 4.4 \times 10^{-40}\ \mathrm{cm}^6\ \mathrm{molecule}^{-2}\ \mathrm{s}^{-1}$$

$$k_2 \leq 1 \times 10^{-20}\ \mathrm{cm}^3\ \mathrm{molecule}^{-1}\ \mathrm{s}^{-1}$$

which are more than 100 times slower than those of Chan et al. (1967). Thus, homogeneous (gas phase) formation of nitrous acid seems too slow to account for HONO formation in the studies of Hanst at al. (1977) and Grosjean and Pitts (1977,1978), and heterogeneous formation (being itself very slow) may account for the low nitrosamine yields reported by these authors. Pitts et al. (1978) also note that the observed formation of nitrosamines from tertiary amines, for which no gas phase mechanism could be proposed, may be entirely heterogeneous. Such processes for the formation of nitrosamines from tertiary amines have been well documented in the bulk (liquid) phase (Ohshima and Kawabata, 1978; Smith and Loeppky, 1967).

Finally, it should be noted that the accepted mechanism for the liquid phase nitrosation of secondary amines involves N_2O_3 (Mirvish, 1975; Ridd, 1961; Scanlan, 1975) according to the reactions:

$$2\ \mathrm{HONO} \rightleftarrows N_2O_3 + H_2O \qquad (6\text{-}57)$$

$$N_2O_3 + R_2NH \rightarrow R_2NNO + \mathrm{HONO} \qquad (6\text{-}58)$$

and that the corresponding rate of nitrosation, $r = k\ (\mathrm{amine})\ (\mathrm{HONO})^2$, may apply to the studies of Hanst and of Pitts and Grosjean in which HONO formation seems to be controlled by heterogeneous processes. The mechanisms of Gehlert and Rolle (1977) and Neurath et al. (1976) for the gas phase reaction also involve N_2O_3 (or NO and NO_2). Furthermore, Hanst et al. (1977) reported that the amine disappearance rate in a mixture containing 1 ppm dimethylamine, 4 ppm NO, and 1 ppm NO_2 in dry nitrogen (i.e., under conditions not conducive to the formation of nitrous acid) was comparable to that measured in the amine-HONO-NO_x-water mixture in air (1 percent min^{-1} and 4 percent min^{-1}, respectively). Thus, the conflicting evidence currently available does not permit firm conclusions regarding the rates, yields and mechanisms of nitrosamine formation from amines and oxides of nitrogen in the dark.

6.4.2 Photochemical Reactions of Amines

In the smog chamber experiments described in the previous section (Grosjean et al., 1977; Pitts et al., 1978), amine-NO_x-air mixtures were also exposed to sunlight for ~2 hours. Diethyl-and triethylamine reacted rapidly to form ozone, peroxyacetylnitrate (PAN) and acetaldehyde as the major gas phase products, as well as light scattering aerosols consisting essentially of the amine nitrates. Several other products were formed in the gas phase including diethylnitramine [$(C_2H_5)_2NNO_2$] and several ethyl substituted amides. These products and the corresponding yields are listed in Table 6-4.

Irradiation of dimethylamine and trimethylamine under the same conditions yielded ozone, formaldehyde, dimethylnitramine [$(CH_3)_2NNO_2$] and several methyl substituted amides in the gas phase as well as aerosol products (Pitts et al., 1978). Reaction products of dimethylamine that have also been identified by other investigators include formaldehyde (Dushumin and

TABLE 6-4. MAXIMUM CONCENTRATIONS AND YIELDS OF THE PRODUCTS OF DIETHYLAMINE AND TRIETHYLAMINE (GROSJEAN ET AL., 1977; PITTS ET AL., 1978)

		From $(C_2H_5)_2NH$			From $(C_2H_5)_3N$		
		Maximum Concentration		Molar Conversion Yield, %[a]	Maximum Concentration		Molar Conversion Yield, %[a]
Product	Formula	µg/m^3	ppb		µg/m^3	ppb	
GAS PHASE							
Ozone	O_3		290			260	
Acetaldehyde	CH_3CHO		300	30[b]		700	47[b]
PAN	$CH_3CO\text{-}OONO_2$		41	4[b]		72	5[b]
GAS PHASE (by GC-MS)							
Dark							
Diethylnitrosamine[c]	$(C_2H_5)_2NNO$	59	14	2.8	17	4.1	0.8
Sunlight							
Diethylnitrosamine[c]	$(C_2H_5)_2NNO$	(destroyed)			38	9.1	1.8
Diethylnitramine	$(C_2H_5)_2NNO_2$	780	162	32	177	37	7.4
Diethylformamide	$(C_2H_5)_2NCHO$	29	7.0	1.4	178	43	8.6
Diethylacetamide	$(C_2H_5)_2NCOCH_3$	3.6	0.8	0.2	15	3.2	0.6
Ethylacetamide	$C_2H_5NHCOCH_3$	42	12	2.4	48	13	2.6
Unidentified, MW=87[d]		--	--	--	41	12	2.4
Diacetamide	$(CH_3CO)_2NH$	--	--	--	trace		

(continued)

TABLE 6-4. (continued)

		From $(C_2H_5)_2NH$			From $(C_2H_5)_3N$		
Product	Formula	Maximum Concentration µg/m³	ppb	Molar Conversion Yield, %[a]	Maximum Concentration µg/m³	ppb	Molar Conversion Yield, %[a]
AEROSOL PHASE[e]							
Sunlight							
b_{scat} (maximum value)		$4 \times 10^{-4} m^{-1}$			$46 \times 10^{-4} m^{-1}$		
TSP		60			370		
Acetamide	CH_3CONH_2	3		0.2	8.7		0.7
Diethylhydroxylamine	$(C_2H_5)_2NOH$	--			7.6		0.4
Nitrates	NO_3^-	42			158		

[a] Initial amine concentrations = 0.5 ppm (calculated from amount injected).

[b] Taking into account the number of ethyl groups in DEA and TEA.

[c] Not corrected for artifact formation (maximum ~ 10% of the observed concentration).

[d] Assuming same mass spectrometer response as diethylacetamide.

[e] Based on volumes sampled: 27.9 m^3 (DEA) and 30.8 m^3 (TEA).

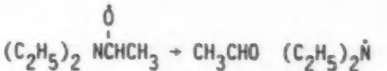
Sopach, 1976; Hanst et al., 1977), dimethylnitramine (Dushumin and Sopach, 1976; Tuazon et al., 1978), and the amine nitrate aerosol (Dushumin and Sopach, 1976; Gehlert and Rolle, 1977).

In the experiments conducted with secondary amines (diethyl and dimethyl), the nitrosamine formed in the dark was progressively destroyed in sunlight, as was reported before for dimethylnitrosamine (Bretschneider and Matz, 1973,1976; Hanst et al., 1977) and diethylnitrosamine (Bretschneider and Matz, 1973,1976). In contrast, the concentration of diethylnitrosamine formed in the dark from the tertiary amine, triethylamine, increased upon irradiation for ~ 60 minutes, reaching a level about 3 times its average concentration in the dark prior to being destroyed upon further exposure to sunlight (Figure 6-3).

The mechanism proposed by the authors (Grosjean et al., 1977; Pitts et al., 1978) involves hydroxyl radical (OH) abstraction on a secondary C-H bond to produce an alkyl radical, as shown here for triethylamine:

$$(C_2H_5)_3N + OH \rightarrow (C_2H_5)_2N\dot{C}HCH_3 \qquad (6\text{-}59)$$

followed by the well-known sequence $\dot{R} + O \rightarrow R\dot{O}$, $R\dot{O} + NO \rightarrow NO + R\dot{O}$ (Demerjian et al., 1974. The alkoxy radical $R\dot{O}$ then decomposes to give two of the major products, acetaldehyde and diethylacetamide:



$$(C_2H_5)_2\ N\overset{\dot{O}}{\dot{C}}HCH_3 \rightarrow CH_3CHO\ \ (C_2H_5)_2\dot{N} \qquad (6\text{-}60)$$

$$\rightarrow (C_2H_5)_2NCHO + \dot{C}H_3 \qquad (6\text{-}61)$$

Further reactions of acetaldehyde lead to PAN, another major product. The diethylamino radical, $(C_2H_5)_2\dot{N}$, reacts with NO and NO_2 to form diethylnitrosamine and diethylnitramine, respectively:

$$(C_2H_5)_2\dot{N}\ \ NO \rightarrow (C_2H_5)_2NNO \qquad (6\text{-}62)$$

$$(C_2H_5)_2\dot{N}\ \ NO_2 \rightarrow (C_2H_5)_2NNO_2 \qquad (6\text{-}63)$$

It is assumed, by analogy with the simplest dialkylamino radical, NH_2, that reaction of $(C_2H_5)_2\dot{N}$ with oxygen is very slow [this has received confirmation very recently in the case of NH_2 (Lesclaux and Demissy, 1978)].

A recent study by Calvert et al. (1978) of the photolysis of dimethylnitrosamine has shown that the dimethylamine radical, $(CH_3)_2\dot{N}$, can react with NO almost 10^6 times faster than with O_2 and with NO_2 approximately 10^7 times faster than with O_2. Nitrous acid and $CH_3N{=}CH_2$ were also identified as major products. These results suggest that dimethylamine radicals formed in a $NO\text{-}NO_2$-polluted atmosphere have a good chance of forming nitrosamines and nitramines even though the concentrations of NO and NO_2 are very small when compared to the amount of molecular oxygen present.

In the case of the secondary amine, diethylamine, the larger nitramine yield indicates that the diethylamine radical is also produced by other reactions, including OH abstraction on the N-H bond:

$$(C_2H_5)_2NH + OH \rightarrow (C_2H_5)_2\dot{N} + H_2O \qquad (6\text{-}64)$$

Rate constants for the OH-amine reaction have been measured recently (Atkinson et al., 1977,1978) and are consistent with both N-H and C-H abstraction. Alkylamines react quite

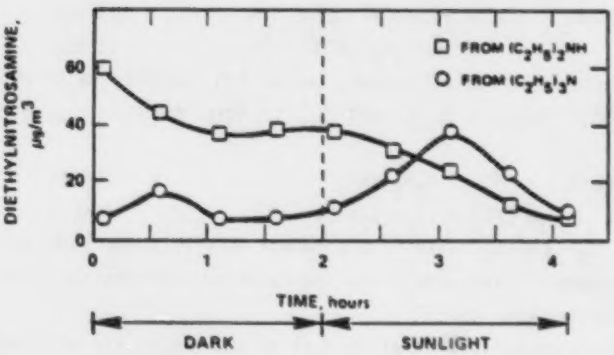


Figure 6-3. Formation and decay of diethylnitrosamine, in the dark and in sunlight, from diethylnitrosamine (open squares) and from triethylamine (open circles)(Pitts et al., 1978).

rapidly with OH, with atmospheric half-lives of 2-3 hours at typical OH concentrations in the lower troposphere (Atkinson et al., 1978).

The efficient formation and accumulation of nitramines (reaction 6-63) is due to their stability in sunlight (Grosjean et al., 1977; Pitts et al., 1978). In contrast, nitrosamines photodecompose readily:

$$R_2NNO \xrightarrow{h\nu} R_2\dot{N} + NO \qquad (6\text{-}65)$$

and their concentration in sunlight is dicated by the competing reactions 6-62 and 6-65. Results shown in Figure 6-3 for triethylamine indicate that photochemical formation (reaction 6-62) may prevail upon photodecomposition during daytime under certain conditions, in contradiction with the generally accepted idea that any nitrosamine present at night in the atmosphere should be rapidly destroyed after sunrise (U.S. EPA, 1976b; Hanst et al., 1977). Processes other than photodecomposition, such as direct oxidation of nitrosamines to nitramines by ozone or other oxidizing species, have not been investigated.

6.4.3 Formation of Nitrosamine in Atmospheric Aerosols

Heterogeneous formation in acid aerosols has been suggested (U.S. EPA, 1976a) as a possible mechanism for nitrosamine production in the atmosphere. The absorption of basic amines by acidic aerosol droplets (containing sulfuric acid and/or ammonium bisulfate), followed by reaction with nitrite, nitrous acid, or other species, could theoretically lead to the formation of nitrosamines. The acid media would favor nitrosamine formation (Mirvish, 1975; Ridd, 1961; Scanlan, 1975) and would prevent rapid photodecomposition during daylight hours since the absorption of nitrosamines is greatly attenuated in acid solutions (Chow et al., 1972).

This hypothetical mechanism, which is intuitively plausible, warrants several comments. First, basic species, such as ammonia, which are present in ambient air at higher concentrations than amines, may compete effectively for absorption in acidic aerosol droplets. Second, nitrosamines photolyze readily in aqueous solutions (Chow et al., 1972; Polo and Chow, 1976) and atmospheric aerosols seldom achieve the high acidity ($pH \sim 1$) necessary to prevent photodecomposition. It should be pointed out, however, that acidic aerosols are not necessarily required, since several studies have shown that nitrosation proceeds quite effectively at neutral and/or alkaline pH by free radical processes (Challis and Kyrtopoulos, 1977) or due to catalysis by carbonyl compounds or metal ions (Keefer, 1976; Keefer and Roller, 1973). Finally, irrespective of the aerosol acidity, reactions of nitrosamine with oxidizing species in aqueous aerosol droplets may lead to the formation of, for example, nitramines.

6.4.4 Environmental Implications

Of the three mechanisms discussed above, nitrosation in atmospheric aerosols is purely speculative at this time. The two other processes involve photochemical and non-photochemical reactions of amines with oxides of nitrogen and related species and may be relevant to the formation of nitrosamines in the atmosphere.

Nighttime production of nitrosamines: Conflicting results are presented in the literature concerning nitrosamine formation rates and yields from secondary and tertiary amines. Several investigators report low yields (a few percent), essentially controlled by the slow rate of nitrous acid formation through heterogeneous processes, while others report high yields achieved within minutes.

Nitrosamines have been shown to form from secondary (Grosjean et al., 1977; Hanst et al., 1977; Pitts et al., 1978) and tertiary amines (Grosjean et al., 1977; Pitts et al., 1978) under simulated atmospheric conditions. Primary amines have not been investigated but should receive some attention since they have been shown to produce nitrosamines, albeit in low yields, in the liquid phase (Wartheson et al., 1975).

If one accepts the low yields of Hanst et al. (1977), Grosjean et al. (1977), and Pitts et al. (1978), nighttime concentrations of nitrosamines in typical urban atmospheric conditions should be quite low. However, caution should be exercised when extrapolating these laboratory and smog chamber data to the ambient atmosphere.

Photochemical reactions of amines: Secondary and tertiary amines react readily with the hydroxyl radical to form aldehydes, PAN, ozone, nitramines, several amides, and the amine nitrate aerosol. Nitrosamines are also formed but photodecompose rapidly. Little is known about ambient levels of amines, but they are presumably low ($\leq$ 10 ppb), and daytime nitrosamine levels should be quite low due to their rapid photodecomposition. However, photochemical formation of diethylnitrosamine from the tertiary amine, triethylamine, has been shown to prevail over photodecomposition for ~ 1 hour in full sunlight (maximum yield ~ 2 percent).

Products other than nitrosamines, i.e., nitramines and amides, may represent health hazards and may warrant further investigation. In industrial environments where, for example, 50-500 ppb of amine might be released into polluted urban air, nitramines (10-30 percent yield, or 5 to 150 ppb) and amides (5-15 percent, or 0.5 to 75 ppb) may form in sunlight. Dimethylnitramine (Druckery et al., 1961; Goodall and Kennedy, 1976) and acetamide (Jackson and Dessau, 1961; Weisburger et al., 1969) are carcinogenic, while dimethylformamide has been identified in urban air (Pellizzari, 1977) and has been shown to undergo nitrosation (Lijinsky et al., 1972; Walker et al., 1976). Another amide, N,N-dimethyl-acetamide, has been identified in diesel crankcase emissions (Southwest Research Institute, 1977).

6.5 SUMMARY

6.5.1 Chemistry of Oxides of Nitrogen in the Lower Atmosphere

Nitrogen oxides undergo many reactions in the lower atmosphere. Triggered by solar radiation, photochemical reactions involving nitrogen oxides and other compounds, principally gaseous organic molecules, result in formation of reactive species capable of initiating a large number of subsequent reactions. In particular, although anthropogenic emissions of NO_x occur principally as NO, atmospheric reactions may produce the more toxic and irritating compound NO_2, which is of direct concern to human health.

The reactive species, which include a variety of unstable, excited molecules and molecular fragments having only transitory existence and/or occurring only in extremely low concentrations, are the principal agents through which chemical changes occur in the polluted urban atmosphere. Many of the reaction sequences involving these unstable or short-lived intermediates are complex in nature, leading inevitably to short-term variations in nitrogen dioxide concentrations, depending upon a variety of factors such as radiant energy input, temperature, and the presence or absence of a variety of hydrocarbons. The dependence of NO_2 concentrations on hydrocarbons results in a coupling between the chemistry of the oxides of nitrogen and the photochemical oxidants, causing atmospheric concentrations of either type of pollutant to depend, to some extent, on atmospheric concentrations of the other. There is considerable current research on the precise relationships among ambient concentrations of NO_x and the photochemical oxidants.

Most of the current knowledge both of atmospheric chemical pathways and of the end products of these reactions rests on controlled experiments conducted in small-scale laboratory smog chambers. It is believed that the chemical processes that take place in these chambers are similar to those that take place in the atmosphere. It is important to note, however, that our current understanding of nitrogen chemistry is not complete and that reaction details and rate constants may be subject to change or new reactions of substantial importance may remain to be discovered.

A number of smog chamber studies using simulated urban atmospheres have been conducted in order to define the relationship between levels of NO_x and hydrocarbon inputs and resulting NO_2 concentrations. The results show that, with other factors held constant, both average and maximum NO_2 concentrations tend to be proportional to initial NO_x inputs. While some disagreement is reported from different chamber studies on the precise effect of hydrocarbon reduction on NO_2 concentrations, a consensus would seem to be as follows: Fifty percent hydrocarbon reduction would have little effect on average NO_2 concentrations (a change of ± 10 percent) but would yield moderate decreases in maximal NO_2 (a reduction of about 10 to 20 percent). It should be noted that these conclusions are meant to apply to one basic type of ambient situation--the situation of well-mixed urban air.

6.5.2 Nitrate and Nitrite Formation

In experimental simulations of a daily cycle of polluted atmospheres, peroxyacetyl nitrate (PAN), known to be very toxic to plants, may be the conversion product of up to one-half of the oxides of nitrogen. PAN, however, is not the final product of these gas-phase reactions, since it may decompose. The most likely final gaseous product is nitric acid, a strong acid and a powerful oxidizing agent. Photochemical models of diurnal atmospheric reactions predict that up to one-half of the original oxides of nitrogen is converted to nitric acid and nitrates. It is believed that nitrates are formed when nitric acid vapor reacts rapidly with ambient ammonia to form small solid particles of ammonium nitrate. Another possible mechanism resulting in aerosol formation consists of the direct absorption of NO_x into aqueous droplets in the presence of ambient ammonia. The relative importance of these two mechanisms is not presently known.

Nitric acid produced in the atmosphere is an important component of acidic rain. Particulate nitrates may be of concern as a component of respirable particulate matter. Both nitric acid and particulate nitrates are of concern for adverse effects on human health.

6.5.3 Transport and Removal of Nitrogenous Species

Over travel distances measured in hundreds of kilometers, more than half the total mass of most pollutants, including nitrogenous species, may be removed from the atmosphere by a variety of processes. These processes are usually grouped into the two generic classes, dry and wet deposition. Gaseous nitrogenous species may be removed by surface absorption (dry deposition). Vegetation and soil are capable of removing significant amounts of NO and NO_2 from the atmosphere by this mechanism. Dry deposition of particulate nitrogenous species may occur through sedimentation, Brownian diffusion, and impaction. The rate or removal by these mechanisms is strongly dependent on wind speed and the detailed properties of the deposition surface. Rainout and washout are two wet deposition mechanisms by which nitrogenous gases and particulates may be removed from the atmosphere. Rainout refers to removal processes taking place within a cloud; washout refers to removal of aerosols and gases below the cloud layer by precipitation. The uptake of NO_x by rain depends upon such parameters as rainfall intensity, raindrop size, and the chemical composition of the droplets. To date, the detailed processes of NO_x removal have not been thoroughly studied.

6.5.4 Mechanisms of Atmospheric Nitrosamine Formation

Three mechanisms possibly leading to the atmospheric formation of nitrosamines and related compounds are as follows:

- Non-photochemical reactions of gaseous amines with oxides of nitrogen and nitrous acid
- Photochemical reactions of amines with oxides of nitrogen in the gas phase
- Heterogeneous nitrosamine formation processes in atmospheric aerosols.

The first two processes have been the object of recent experimental studies, including simulation experiments in environmental chambers. The third process involving aerosol particles is purely speculative at this time.

Conflicting results are presented in the literature concerning nitrosamine formation rates and yields from secondary and tertiary amines. Several investigators report low yields (a few percent) essentially controlled by the slow rate of nitrous acid formation through heterogeneous processes, while others report high yields achieved within minutes.

Nitrosamines have been shown to form from secondary and tertiary amines under simulated atmospheric conditions. Primary amines have not been investiaged. If one accepts the lowest yields reported, nighttime concentrations of nitrosamines under typical urban conditions would not be significant. However, caution should be exercised when extrapolating these laboratory and smog chamber data to the ambient atmosphere, especially in view of the lack of consensus on formation rates.

In sunlight-irradiated atmospheres, secondary and tertiary amines react readily with the hydroxyl radical to form aldehydes, PAN, ozone, nitramines, several amides, and amine nitrate aerosol. Nitrosamines are also formed but photodecompose rapidly. Little is known about ambient levels of amines, but they are presumably low ($\leq$ 10 ppb), and daytime nitrosamine levels should be quite low due to their rapid photodecomposition. However, photochemical formation of diethylnitrosamine from the tertiary amine, triethylamine, has been shown to prevail over photodecomposition for ~ 1 hour in full sunlight (maximum yield ~ 2 percent).

Products other than nitrosamines, i.e., nitramines and amides, may represent health hazards and may warrant further investigation. Near certain industrial environments where, for example, 50-500 ppb of amine might be released into polluted urban air, nitramines (10-30 percent yield or 5 to 150 ppb) and amides (5-15 percent, or 0.5 to 75 ppb) may form in sunlight.

6.5.5 Source-Receptor Relationships

The question of predicting the NO_2 concentration experienced by a receptor, such as a human being, due to given emissions of NO_x has received little attention in the literature until very recently. Relationships among ambient NO_x, hydrocarbons, and ozone which make use of the complex chemistry described above have been considered in connection with the question of predicting ambient ozone concentrations. Some of the methodology (which includes a variety of air quality simulation models) applicable to the ozone problem has potential application to the case of NO_x source-receptor relationships. However, neither development nor validation of these models for the NO_2 problem has reached a stage where it is possible to evaluate with any certainty either their usefulness or accuracy. For this reason, no general statements can prudently be made at this time concerning the specific impacts on air quality of various NO_x source types. Such considerations must await documentation of adequate modelling procedures.

CHAPTER 6

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7. SAMPLING AND ANALYSIS FOR AMBIENT NO_x AND NO_x-DERIVED POLLUTANTS

7.1 INTRODUCTION

This chapter summarizes a variety of methods used for measuring oxides of nitrogen (NO_x) and other pollutants which may be derived from NO_x through atmospheric transformations (Chapter 6). Emphasis is placed upon describing methodology currently available or in general use for routine monitoring of ambient pollutant concentrations.

An appreciation of some of the errors involved in present monitoring techniques is important in evaluating the quality of ambient pollution data. Three types of errors are discussed in this section: interferences, systematic errors and random errors.

The measurement of individual pollutants in ambient air is complicated by the presence of other airborne chemicals which may produce responses in the measuring apparatus generally indistinguishable from those produced by the pollutant being monitored. These spurious responses are known as "interferences." Extensive tests are conducted by the U.S. Environmental Protection Agency (U.S. EPA) and/or other laboratories on potential interferences in proposed measurement techniques before they are considered suitable for routine monitoring. This chapter describes reported interferences for the methods listed. It should be noted, however, that not all potential interferences have equal significance. Their magnitude will, in general, depend on the ambient concentrations of the interfering species, the inherent sensitivity of a given procedure to spurious responses, and, in some cases, on details of the measuring apparatus which may vary from instrument to instrument. An analytic technique sensitive to interference may still be useful if the interfering species occurs only in low concentrations in ambient air or may otherwise be accounted for.

In addition to errors introduced by interferences, a given analytic technique may be subject to systematic over- or underestimation of the pollutant concentration which affects the accuracy with which these concentrations are known. Such errors are known as "biases." The assessment of the magnitude of such biases for a given analytical method generally requires extensive testing by a number of laboratories sampling the same pollutant concentration in ambient air (collaborative testing).

Random errors introduced by unknown factors such as variability in detailed procedures used by different operators or sensitivity of the method to small uncontrollable variations in operational parameters are generally known collectively as an imprecision in the method. A measure of this type of error often used is the standard deviation of a set of measurements. Once the standard deviation is known, it may be expected on statistical grounds that about 95 percent of measurements made on the same ambient air sample will yield values for the NO_2 concentration which differ from the average by at most $\pm$ 2 standard deviations. The precision of a method is also often assessed in collaborative testing procedures. The results of testing for these two error types are described in this chapter where available.

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A critical analysis of techniques for measuring nitrogen oxides in ambient air has been reported very recently by Saltzman (1979). In addition, a historical review of USEPA NO_2 monitoring methodology requirements has been given by Purdue and Hauser (1979).

In considering the analytic techniques described below it must be noted that the state-of-the-art of measurement technology is constantly changing. For this reason, techniques currently in use or recommended for use may be replaced by better methods at some future time, techniques which are presently in the development stage may become routine in the future, or entirely new techniques may be developed.

Since the publication in 1971 of the original document Air Quality Criteria for Nitrogen Oxides, there have been significant changes in the technology associated with measurement of ambient concentrations of both NO and NO_2. In addition, concern about the potential adverse human health implications of ambient concentrations of other NO_x-derived compounds such as suspended nitrates, nitric acid, and N-nitroso compounds has led both to development of new analytic techniques and to a reexamination of existing methodology for their measurement.

With regard to the measurement of NO_2, the original Federal Reference Method, the Jacobs-Hochheiser technique, was discovered to have unresolvable technical difficulties. The USEPA published the following brief summary of these difficulties on June 8, 1973 when it withdrew the method (U.S. EPA, 1973):

> ". . . EPA's analysis indicates that the reference method is deficient in two aspects. First, the method overestimates nitrogen dioxide concentrations at low levels and underestimates them at high levels because the collection efficiency of the absorbing reagent is dependent upon nitrogen dioxide concentration being measured. Second, the method is subject to positive interference by nitric oxide. Since the variable collection efficiency problem cannot be resolved, this method can no longer serve as the reference method."

After extensive testing, the USEPA promulgated, on December 1, 1976, the chemiluminescence measurement principle and associated calibration procedures (U.S. EPA, 1976) upon which a number of chemiluminescent analytical instruments are based. These analyzers, once approved by the U.S. EPA (1975a) are referred to as Reference Methods. For purpose of simplicity in the descriptions to follow, however, the term Reference Method is meant to apply both to the measurement principle and to the instruments based thereupon. The required performance specifications which acceptable continuous chemiluminescence analyzers must meet are shown in Table 7-1.

Equivalent methods are methods based on measurement principles different from the reference method. Two kinds of equivalent methods are possible--manual and automated (continuous monitoring analyzers). Candidate automated methods may be designated as equivalent methods if they meet the performance specifications listed in Table 7-1 and demonstrate a

TABLE 7-1. PERFORMANCE SPECIFICATIONS FOR NITROGEN DIOXIDE AUTOMATED METHODS (U.S. EPA, 1976b)

Performance Parameter	Units	Nitrogen Dioxide
1. Range	ppm	0-0.5
2. Noise	ppm	0.005
3. Lower detectable limit	ppm	0.01
4. Interference equivalent		
Each interferant	ppm	±0.02
Total interferant	ppm	0.04
5. Zero drift, 12 and 24 hr	ppm	±0.02
6. Span drift, 24 hr		
20 percent of upper range limit	%	±20.0
80 percent of upper range limit	%	±5.0
7. Lag time	minutes	20.0
8. Rise time	minutes	15.0
9. Fall time	minutes	15.0
10. Precision		
20 percent of upper range limit	ppm	0.02
80 percent of upper range limit	ppm	0.03

consistent relationship to the reference method during side-by-side ambient monitoring. Candidate manual methods need only demonstrate a consistent relationship to the reference method to be designated as equivalent methods. Table 7-2 shows the test specifications which must be met to demonstrate a consistent relationship with the reference method.

TABLE 7-2. CONSISTENT RELATIONSHIP TEST SPECIFICATIONS FOR NITROGEN DIOXIDE (U.S. EPA, 1976b)

Concentration Range, ppm NO_2	Maximum Discrepancy Specification, ppm
Low 0.02 to 0.08	0.02
Medium 0.10 to 0.20	0.02
High 0.25 to 0.35	0.03

In addition to the Reference Method, several other methods, notably those designated in SAROAD (see Chapter 8) as the instrumental colorimetric Griess-Saltzman method, the Lyshkow-modified Griess-Saltzman method, the triethanolamine method, the sodium arsenite method, and the TGS-ANSA method have also been extensively tested and are currently in widespread use and/or have been extensively tested, and are available for routine measurement of ambient NO_2 concentrations. Both the sodium arsenite method and the TGS-ANSA method were designated as equivalent methods as of December 14, 1977. This means data obtained by these two methods are accepted by EPA as equivalent to chemiluminescence data (Reference Method) for the purpose of establishing attainment status with respect to the National Ambient Air Quality Standard (NAAQS) for NO_x. The manual Griess-Saltzman method, although not usually used in ambient monitoring, is discussed because a number of studies pertinent to an assessment of the health effects have used this method in laboratory situations (Chapters 14 and 15).

Although adequate chemical techniques exist for the determination of the nitrate fraction of suspended particulate matter in ambient air, a number of very recent reports have pointed to significant nitrate artifact formation on the glass fiber filters in widespread use for collecting the particulate matter. At this time, therefore, most of the existing data base on urban ambient nitrate concentrations must be considered to be of doubtful validity. Since consistent relationship to the reference method during side-by-side ambient monitoring. Candidate manual methods need only demonstrate a consistent relationship to the reference method to be designated as equivalent methods. Table 7-2 shows the test specifications which must be met to demonstrate a consistent relationship with the reference method. positive artifact formation has been shown to be associated with conversion of ambient NO_2 and/or nitric acid (HNO_3) to nitrates and since sulfuric acid aerosol has been implicated in removal of nitrate, data from certain background sites may be validated in special cases where it can be shown that the concentrations of these species were sufficiently low during the monitoring period of interest.

Recent discovery of N-nitroso compounds in food, water, and ambient air has prompted the development of a variety of new instrumental techniques in the last few years. Measurement technology is still developing and insufficient time has elapsed for careful evaluation of existing techniques. In particular, some difficulties have been reported to be associated with artifact formation under certain sampling conditions.

Development of long-pathlength infrared absorption techniques has recently made possible the observation of nitric acid in ambient air. However, the technique is presently too elaborate for routine monitoring applications. Other techniques for quantitative analysis of nitric acid vapor have been reported but have not yet been carefully evaluated.

The sections in this chapter describing briefly the analysis of nitrate in media other than air (e.g., water, soil, and plant and animal tissue) are included for two reasons: (1) the basic methodology is similar to that used for analyzing aqueous extracts of nitrate particulate matter drawn from the ambient air and (2) it is believed appropriate to make some estimate of human exposure from different media with a view towards placing exposure via the atmospheric route in perspective. Similar considerations prompted the inclusion of material on techniques for measuring N-nitroso compounds in food and water.

7.2 ANALYTICAL METHODS FOR NO_x

Many methods have been used to measure NO_x concentrations in air. Some of these methods directly measure the species of interest; others require that the species be oxidized or reduced, or separated from interferences before the measurement is made. Of particular importance in this regard is the new Reference Method, the continuous chemiluminescence method. The method is specific for NO but may easily be modified for measurement of NO_2 by first quantitatively reducing the NO_2 to NO. The regulatory specifications relating to the Reference Method (RM) are prescribed in Title 40 of the Code of Federal Regulations, Part 50, Appendix F.

7.2.1 The Reference Method for NO_2: Gas-Phase Chemiluminescence

Atmospheric concentrations are measured indirectly by first reducing the NO_2 quantitatively to NO, then reacting the resultant NO with O_3 and measuring the light intensity from the reaction.

The use of the gas-phase chemiluminescent reaction of NO and O_3 for quantitative measurement of NO was reported initially by Fontijn et al. (1970) and some improvements were described by Stedman et al. (1972). The sample air stream is mixed with air containing a high O_3 concentration (approximately 1 percent). The reaction of NO and O_3 forms excited NO_2 molecules, the number of which is proportional to the NO concentration. Some of the excited NO_2 molecules emit electromagnetic radiation with wavelengths between 600 and 2000 nm, with a maximum at 1200 nm (Clough and Thrush, 1967). The reaction chamber is held at reduced pressures to decrease the collisional deactivation, and the emitted radiation is measured with a photomultiplier tube and associated electronics. To reduce interferences of the chemiluminescent reactions of ozone with other species, optical filters can be employed (Stevens and Hodgeson, 1973). Typical commercial chemiluminescence instruments can detect concentrations as low as 9.5 $\mu g/m^3$ (0.005 ppm) (Katz, 1976).

Since the detection of NO_2 by the RM is directly dependent on the analyzer's capability to reduce NO_2 to NO, it is important that the conversion be essentially quantitative over a wide range of NO_2 concentrations.

Catalytic reduction of NO_2 to NO is commonly employed in chemiluminescence NO-NO_x instruments. These instruments measure NO alone by passing the sample directly to the detector. The total concentration of NO and NO_2 (NO_x) is measured by drawing the sample through a catalytic reduction unit prior to entering the detector. NO_2 concentrations are obtained by subtraction.

Winer et al. (1974) studied the reactions of various nitrogen compounds over carbon and molybdenum converters. It was found that not only NO_2 but peroxyacetyl nitrate (PAN) and a wide variety of organic nitrogen compounds were reduced to NO quantitatively; nitroethane and nitric acid were partially reduced. Joshi and Bufalini (1978) report non-quantitative positive inferences from halocarbons in commercial instruments using a heated carbon converter. They also speculate that instruments using high temperature stainless steel converters may be subject to interferences from chlorinated hydrocarbons. The authors suggest replacing heated carbon converters with $FeSO_4$ converters; however, this technique has not been thoroughly evaluated. There is also evidence that converters operating at high temperatures may oxidize ammonia (NH_3) to NO (U.S. EPA, 1973). This can be of importance in measuring NO_2 exposures in animal studies, where elevated levels of NH_3 may be present as a result of biologic processes. It is not expected to be of importance in ambient situations using EPA approved analyzers.

While care must be exercised in the use of chemiluminescence instruments because of potential interferences, in most ambient situations NO plus NO_2 are present in much higher concentrations than interfering species.

Results of the USEPA's collaborative quality assurance testing of the method (Constant et al., 1975a) showed that, for one-hour instrumental averaging times for NO_2 concentrations ranging from 50 to 300 $\mu g/m^3$ (0.027 to 0.16 ppm), the method has an average negative bias of five percent with a standard deviation of about 14 percent for equivalent samples measured by different laboratories.

One collaborator had very large biases, and another collaborator had unstable biases. EPA concluded that for most collaborators (8 out of 10), however, the bias is small and well-balanced. The method is satisfactory for averaging times of one hour or more.

7.2.2 Other Analytical Methods for NO_2

7.2.2.1 Griess-Saltzman Method

7.2.2.1.1 General description of method. This chemical method for collection and analysis of NO_2 was originally proposed by Ilosvay (Threadwell and Hall, 1935). Many variations of this method exist, including both manual and automated versions.

The principle of the reaction is thought to be the formation of nitrous acid by the reaction of NO_2 with water. This in turn is reacted with an aromatic amine to form a diazonium salt. In a further step, addition of an organic coupling agent forms a deeply-colored azo dye. The amount of NO_2 collected is related to the light absorbance of the solution.

Many variations of the Griess-Ilosvay reaction have been explored; the variation developed by Saltzman (1954) is one of the most widely used. A manual version of this method has been designated as a Tentative Method by the Intersociety Committee on Methods for Ambient Air Sampling and Analysis (1977c), and was adopted as a standard method by the American Society for Testing and Materials (ASTM, 1974). It has been shown that many different reagent formulations are possible so long as they all contain a diazotizer, a coupler, a buffer, and a surfactant (Kothny and Mueller, 1966).

If an extended sampling time is required in the manual version, the azo dye may suffer bleaching by SO_2. Saltzman (1954) recommended addition of acetone to prevent this. In addition, if the sample is collected in an evacuated bottle or syringe, a long waiting period such as might occur in a manual procedure may cause some NO to be oxidized to NO2 (ASTM, 1974a).

Since the conversion of NO_2 to azo dye is not quantitative, a factor is introduced to represent the conversion efficiency under a given set of experimental conditions. This is often termed the "stoichiometric factor." If the experimental conditions are the same as Saltzman's original formulation (1954) or his modified version (1960) the stoichiometric factor has been reported to be 0.72 (Saltzman, 1954, 1960; Saltzman and Wartburg, 1965; Shaw, 1967). Scaringelli et al. (1970) obtained a value of 0.764. However, recent work by two California groups,[175] the California Air Resources Board (CARB) and the Air and Industrial Hygiene Laboratory (AIHL) has shown that measurements performed by the manual Saltzman technique are sensitive to the exact concentration of the coupling compound NEDA used in the reagent. Careful evaluation showed that values obtained using either the original Saltzman reagent (1954) (0.5 percent sulfanilic acid, 14 percent acetic acid, and .002 percent NEDA) or the modified Saltzman reagent (1960) (0.5 percent sulfanilic acid, 5 percent acetic acid, and .005 percent NEDA) are biased approximately 13 percent too high. The stoichiometric factor is less sensitive to variations in the concentrations of acetic and sulfanilic acids and is independent of relative humidity, temperature of the absorbing agent, and nitrogen dioxide concentration.

7.2.2.1.2 Continuous Saltzman Procedures. In this procedure, NO_2 in ambient air is continuously absorbed in a solution of diazotizing-coupling reagents to form an azo-dye which absorbs light, with a maximum absorbance at approximately 540 nm. The transmittance, which is a function of the NO_2 concentration, is measured continuously in a colorimeter and the output read on a recorder or a digital voltmeter.

The continuous Saltzman procedure currently used in ambient air monitoring has recently been evaluated by EPA (1975b). The results show that static calibration (with solutions prepared to contain known quantities of the nitrite ion) is not uniformly reliable, due to variable collection efficiency of the absorption system. Dynamic calibration procedures by means of a reliable NO_2 permeation device, such as the National Bureau of Standards Standard Reference Material 1629, are recommended since collection efficiency errors and the use of stoichiometric factors are eliminated by virtue of the fact that errors, if they exist, cancel out. Ozone has been reported to be a negative interferent in the continuous version of the method (Baumgardner et al., 1975).

Recently however, Adema (1979) could not confirm the interference in one experimental arrangement of a modified manual method. Two specific variants of the continuous method have been used widely for ambient air monitoring. Although both are continuous colorimetric techniques suitable for averaging times of one hour or more, they differ in the use of two distinct absorbing solutions, in which azo dyes are formed. The first, sometimes known as the instrumental colorimetric Griess-Saltzman method, uses a modification of the originally proposed reagent for manual analysis (Saltzman, 1954) and contains 0.5 percent sulfanilic acid, 5.0 percent acetic acid, and 0.005 percent NEDA (Saltzman, 1960). Interferences by three alkyl nitrites, ethyl, n-butyl, and isoamyl have been reported in a manual procedure using this solution (Thomas et al., 1956). The second method, sometimes known as the Lyshkow-modified Griess-Saltzman method, uses an absorbing solution consisting of 0.15 percent sulfanilamide, 1.5 percent tartaric acid, 0.005 percent N-(1-naphthyl)-ethylenediamine dihydrochloride (NEDA), and 0.005 percent 2-naphthol-3, 6-disulfonic acid disodium salt. Both variants are useful in the range of ambient concentrations from 19 to 9,400 μg/m^3 (0.01 to 5.0 ppm). Results of the USEPA's quality assurance testing over the range 90 to 370 μg/m^3 (0.05 to 0.2 ppm) indicate an average bias of six percent with a 13 percent standard deviation among different laboratories testing equivalent samples (Constant et al., 1975b). Estimates of bias for different laboratories, however, varied considerably.

EPA concluded that the overall average results are reasonably accurate but that the method may produce extremely inaccurate readings in an unpredictable fashion. About half the collaborators did achieve fairly stable results for the experiment, leading EPA to offer the subjective judgment that although the method is difficult to use, it will produce reliable results in some hands.

7.2.2.1.3 Manual Saltzman Procedure. In this method, NO_2 in ambient air is drawn at a known rate through an absorbing solution (Saltzman, 1954) in a fritted-glass bubbler for a specified time period, producing an azo-dye. The absorbance of the solution is subsequently measured manually with a spectrophotometer. The NO_2 concentrations present in ambient air may be related to the absorbance of the azo-dye solution by calibration with other solutions containing known quantities of the nitrite ion.

The manual Saltzman procedure is described in this section because a number of studies bearing on the effects of NO_2 on human health have reported using this procedure (see Chapters 14 and 15). When fritted bubblers are employed, the method has been reported to have a usable range of 10 to 9,400 μg/m^3 (0.005 to 5.0 ppm). A precision of 1 percent of the mean concentration is obtainable (Intersociety Committee, 1977c).

ASTM has evaluated the precision and accuracy of the measurement in ambient situations (1974b). A fritted bubbler, cleaned with dichromate solution, was used exclusively in the evaluation. The nominal flow rate for sampling was 0.4 liter/minute. Ten feet of TFE fluorocarbon tubing having a nominal inside diameter of 8 mm was used as a sample probe prior to the fritted bubbler. The technique used to evaluate the accuracy of the method was to spike ambient air, containing NO_2, with additional, accurately known, concentrations of NO_2 and then to attempt to measure the spiking concentrations. The NO_2 used for spiking was obtained from a permeation tube. Results showed an overall positive bias of 18 percent in measuring the spike concentrations. It should, however, be noted that the bias varied significantly among the seven laboratories participating in the study and was, moreover, dependent on NO_2 concentration.

It should be noted that this evaluation technique subjects the method to possible interferences from other pollutants in ambient air. As such, it may be taken to represent the accuracy of results, in the same NO_2 concentration range, expected in health-related studies which use ambient air. Maximum ambient NO_2 concentrations were about 250 $\mu g/m^3$ during the test and maximum total NO_2 (ambient plus spike) concentrations used in the test were about 400 $\mu g/m^3$. Extrapolation of the test results to situations where high NO_2 levels were measured without dilution must be viewed as speculative.

In the case of studies which are carefully controlled with regard to the occurrence of pollutants other than NO_2, it may be expected that the accuracy of the method would be significantly improved, although a similar interlaboratory test under interference-free conditions is lacking in the literature.

As discussed in Section 7.2.2.1.1, recent measurements have resulted in a redetermination of the stoichiometric factor for the Saltzman technique. These results have possible important implications for the numerical values of effects levels reported both in animal (Chapter 14) and human (Chapter 15) studies. They strongly suggest that results obtained by the manual Saltzman technique and referred to Saltzman's original stoichiometric factor are, in fact, about 14 percent too high, i.e., effects probably occurred at lower levels than those reported.

7.2.2.2 Jacobs-Hochheiser Method--The Jacobs-Hochheiser technique was formerly the Federal Reference Method, but is currently unacceptable for air pollution work for reasons cited above. This technique is discussed here mainly because the method was frequently employed in past years in obtaining data for use in epidemiological studies.

The method was developed by Jacobs and Hochheiser (1958) to avoid the bleaching of the azo dye by SO_2 that occurs in the Griess-Saltzman method. Nitrogen dioxide is absorbed in a solution of sodium hydroxide (NaOH) with butanol added as a surfactant to improve gas transfer when using a fritted bubbler. After sampling, any sulfite from absorbed SO_2 is oxidized with hydrogen peroxide. The nitrite in solution is not affected. The solution may then be stored for 48 hours or more before analysis. To quantitate the nitrite in solution, the solution is first acidified with phosphoric acid. A diazotizer and a coupling agent are then added to produce an azo dye. Solution absorbance is then measured spectrophotometrically. The original method was designed for intermittent 40-minute sampling but was later modified for composite sampling over 24 hours. This method, modified again, was employed by the National Air Sampling Network (NASN) (Morgan et al., 1967a, 1967b) and was adopted as the Federal Reference Method in 1971 (U.S. EPA).

The National Academy of Sciences document on nitrogen oxides (National Research Council, 1977) cites an extensive list of references documenting that the sampling efficiency of the Jacobs-Hochheiser method is affected by sampling flow rate, porosity of bubbler frits, liquid level, sampling-container material, incoming pollutant concentration, and contaminants present in the sample. Sampling efficiencies, in the work reviewed, ranged from 15 to 78 percent when 0.1N NaOH alone was used as an absorbant. In addition to the varying efficiency with which NO_2 is removed from the gas sample, the measurement also is affected by the stoichiometric factor. This factor is also variable and may be affected by the presence of hydrogen donors (Christie et al., 1970; Huygen and Steerman, 1971; Morgan et al., 1967a, 1967b; Nash, 1970). A composite of 11 sites sampled in the NASN network gave an average stoichiometric factor of 62 ± 7 percent (Morgan et al., 1967a). The range of measurable concentrations is related to the percent transmission of azo dye solutions measurable with a spectrophotometer. With 50 ml of absorbing reagent and a sample flow rate of 200 ml/min for 24 hours, the range of the method is 20 to 740 $\mu g/m^3$ (0.01 to 0.4 ppm) NO_2 (Katz, 1976). Katz (1976) reported relative standard deviations of 14.4 and 21.5 percent at NO_2 concentrations of 140 and 200 $\mu g/m^3$ (0.07 and 0.11 ppm), respectively.

Because the sampling efficiency and stoichiometric factor are significantly affected by the details of the method employed, by NO_2 concentration, and by constituents of the sample other than NO_2, the use of many modifications of the Jacobs-Hochheiser method in air quality and epidemiological studies has led to data of questionable quality, or even questionable relative comparability.

7.2.2.3 Triethanolamine Method--The method described by Levaggi et al. (1973) utilizes an absorbing solution of triethanolamine and n-butanol surfactant. After collection, the analysis for nitrite is performed with Griess-Saltzman reagent to produce the azo dye for spectrophotometric measurements. Sampling efficiencies up to 95 to 99 percent have been reported (Intersociety Committee, 1977c; Levaggi et al., 1973). The U.S. EPA has recently reported (Ellis and Margeson, 1974) a laboratory evaluation of the method for each of three absorbing solutions (0.1N TEA with n-butanol added at the concentration levels 3 ml/l, 0.5 ml/l and 0.0 ml/l). The results indicate the collection efficiency to be constant, at approximately 80 percent for the first two solutions (78.8 percent for the last solution), over the range of 30 to 700 $\mu g/m^3$ (0.01 to 0.37 ppm) if glass frits are used. If restricted orifices, which are less fragile and cheaper than glass frits, are used, the collection efficiency falls to about 50 percent. For this reason, the USEPA did not subject the method to collaborative testing so that no reliable measure of accuracy and precision is presently available. No interference is expected from SO_2, O_3, or NO at ambient levels and the sample solutions are stable for three weeks after sampling. The accuracy is considered to be comparable to that of the Griess-Saltzman method (Katz, 1976) with bias errors less than two percent at a stoichiometric factor of 0.764 (Scaringelli et al., 1970). This method is presently considered to be a 24-hour method.

7.2.2.4 Sodium-Arsenite Method--The use of an alkaline solution of sodium arsenite (Na_3AsO_3) to absorb NO_2 was described by Christie et al. (1970) and Merryman et al. (1973). Christie et al. reported a collection efficiency of 95 percent using an orifice bubbler. The USEPA recently has evaluated the sodium-arsenite procedure (Beard and Margeson, 1974) and has designated it an equivalent method as of December 14, 1977. The method is presently considered to be a 24-hour method. The results showed that the procedure has a constant collection efficiency for NO_2 of 82 percent over the recommended useful concentration range, 20 to 750 $\mu g/m^3$ (0.01 to 0.4 ppm). Results of the USEPA's collaborative quality assurance testing of the method indicate a negative bias of three percent with an interlaboratory standard deviation of 11 $\mu g/m^3$ independent of concentration (Constant et al., 1975c). EPA concluded that the measurement errors were essentially uniform for all collaborators, although some dependence on NO_2 level was noted. Eight of ten collaborators exhibited a uniform percent bias over all NO_2 levels tested.

Following absorption, any sulfite is oxidized with peroxide and the solution is then acidified with phosphoric acid. The azo dye is formed by addition of sulfanilamide and N-(1-naphthyl) ethylenediamine dihydrochloride. According to Katz (1976), NO in the air sample can produce a positive interference by increasing the NO_2 response in the sample by 5 to 15 percent of the NO_2 actually present. Carbon dioxide, in excess of typical ambient concentrations, can lead to a negative interference and the method response is affected by sample flow rates in excess of 300 ml/min. The samples are stable for six weeks. Recently, the USEPA also has conducted an evaluation of potential NO and CO_2 interferences (Beard et al., 1975). Results show that, in the range 50 to 310 $\mu g/m^3$ (0.04 to 0.25 ppm) NO and 360,000 to 900,000 $\mu g/m^3$ (200 to 500 ppm) CO_2, the average effect of these interferents is to increase the indicated NO_2 response by 10 $\mu g/m^3$ over the range 50 to 250 $\mu g/m^3$ NO_2 (0.03 to 0.13 ppm).

7.2.2.5 TGS-ANSA Method--A 24-hour manual method for the detection and analysis of NO_2 in ambient air, the TGS-ANSA method was first reported by Mulik et al. (1973). It has been designated an equivalent method to the RM as of December 17, 1977. Ambient air is bubbled, via a restricted orifice, through a solution containing triethanolamine, o-methoxyphenol, and sodium metabisulfite. The NO_2 gas in the ambient sample is converted to nitrite ion (NO_2^-) which is then analyzed by diazotization and coupling using sulfanilamide and the ammonium salt of 8-anilino-1-naphthalene-sulfonic acid (ANSA). The absorbance is read at 550 nm. The function of the triethanolamine is to provide a basic collecting medium (Levaggi et al., 1973). The addition of o-methoxyphenol raises the collection efficiency to 93 percent when using a restricted orifice (Nash, 1970). The sodium metabisulfite inhibits free-radical formation and, hence, the formation of quinones from the o-methoxyphenol as the solution ages.

The collection efficiency for NO_2 is constant at concentrations between 20 and 700 $\mu g/m^3$ (0.01 and 0.37 ppm), which is the range of the method with 50 ml of absorbing reagent and a sampling rate of 200 cm^3/min for 24 hours. No interferences were reported at an NO_2 concentration of 100 $\mu g/m^3$ (0.05 ppm) for the following pollutants at the levels shown in parentheses:

ammonia (205 μg/m^3 or 0.29 ppm); CO (154,000 μg/m^3 or 134 ppm); formaldehyde (750 μg/m^3 or 0.61 ppm); NO (734 μg/m^3 or 0.59 ppm), phenol (150 μg/m^3 or 0.04 ppm); O_3 (400 μg/m^3 or 0.2 ppm); and SO_2 (439 μg/m^3 or 0.15 ppm). The absorbing reagent is stable for three weeks before sampling and the collected samples are stable for three weeks after sampling (Fuerst and Margeson, 1974; Mulik et al., 1973).

Results of USEPA's collaborative quality assurance testing (Constant et al., 1974) indicate a lower detectable limit < 15 μg/m^3 (0.008 ppm), an average bias of 9.5 μg/m^3 (0.005 ppm) over the range of 50-300 μg/m^3 (0.03 to 0.16 ppm), and an interlaboratory standard deviation of 8.8 μg/m^3 (0.005 ppm). EPA concluded that the errors were essentially uniform for all collaborators. The biases shown were nearly independent of the NO_2 level.

7.2.2.6 Other Methods--In addition to the standard wet chemical methods for NO_2 measurement, many other techniques have been explored. Molecular correlation spectrometry compares a molecular absorption band of a sample or plume with the corresponding absorption band of NO_2 stored in the spectrometer (Williams and Kolitz, 1968). Spectrometers processing the second derivative of sample transmissivity with respect to wavelength have been employed to measure NO_2 as well as NO (Hager and Anderson, 1970). Infrared lasers and infrared spectrometry have been applied by Hanst (1970), Hinkley and Kelley (1971) and Kreuzer and Patel (1971).

7.2.2.7 Summary of Accuracy and Precision of NO_2 Measuring Methods--Purdue et al. reported an extensive comparison of both intra- and intermethod accuracy and precision of the chemiluminescent, sodium arsenite, TGS-ANSA and continuous colorimetric (Lyshkow modification) procedures for NO_2 measurement (Purdue et al., 1975). The study was conducted under carefully controlled laboratory conditions using skilled technicians. Ambient air spiked with NO_2 was sampled by two identical instruments for each method tested. In the case of manual methods four samples were taken. The NO_2 spikes were varied randomly from day to day over a 20-day sampling schedule. Spikes ranging from 0 to 800 μg/m^3 were used. The sampling period was of 22 hours duration. Table 7-3 gives the results of a statistical analysis of the intra- and intermethod differences.

Examination of the table reveals that the average difference between any of the methods was never greater than 7.5 μg/m^3 NO_2. The worst case of intramethod differences occurred with the continuous colorimetric method where there was a small bias of 7.5 μg/m^3 NO_2 between the data from the two analyzers. The correlation coefficients for the intermethod comparisons were greater than 0.985 in all cases. In another phase of the study, no intermethod differences could be attributed to concentrations of nitric oxide, carbon dioxide, ozone, total sulfur, or total suspended particulate matter in the ambient air samples. Significant negative interference in the continuous colorimetric method was found at NO_2 concentrations of 75 and 100 μg/m^3 in the presence of ozone at concentrations of 353 and 667 μg/m^3. At an ozone concentration of 100 μg/m^3, no interference was detected. At NO concentrations as high as 302 μg/m^3 no interference was found in the sodium arsenite procedure, although NO has been cited as a positive interferent in the method.

TABLE 7-3. STATISTICAL ANALYSIS OF NO_2 MEASURING METHOD DIFFERENCE[a] ($\mu g/m^3$)

	Sampling Spiked Ambient Air					
Comparison	Pairs	Mean[b] ($\mu g/m^3$)	Standard Dev.[c]	95% C.I.[d] Lower	95% C.I.[d] Upper	Corr. Coeff.
Intramethod						
Chemil/Chemil	22	1.3	5.6	-1.1	3.3	0.999
Color/Color	20	7.5	7.5	3.8	11.3	0.995
ARS/ARS (A)	22	0.6	5.6	-1.9	+3.8	0.997
ARS/ARS (B)	22	0.2	5.6	-1.9	1.9	0.996
TGS/TGS (A)	20	-0.9	3.8	-1.9	0.8	0.999
TGS/TGS (B)	22	0.2	9.4	-3.8	+3.8	0.992
ARS (A)/ARS (B)	22	-2.6	5.6	-5.6	+0.1	0.997
RGS (A)/TGS (B)	20	0.6	7.6	-3.8	+3.8	0.996
Intermethod						
Chemil/Color	20	3.8	7.5	0.0	+7.5	0.994
Chemil/ARS	22	-1.9	9.4	-5.6	+1.9	0.991
Chemil/TGS	20	5.6	9.4	+0.9	9.4	0.990
Color/ARS	20	-3.8	11.3	-9.4	1.5	0.989
Color/TGS	18	3.8	11.3	-1.9	9.4	0.985
ARS/TGS	20	7.5	7.5	+3.8	11.3	0.994

[a] Extracted from Purdue et al., 1975.

[b] Signed difference.

[c] Standard deviation.

[d] 95 percent confidence interval of mean difference.

[e] Correlation coefficient between paired values in calculating mean difference.

A summary of available reliability estimates derived from collaborative testing studies is given in Table 7-4 for four ambient air monitoring procedures: Chemiluminescence, Griess-Saltzman (continuous colorimetric using dynamic calibration), TGS-ANSA, and sodium arsenite. Measures of accuracy (bias) and precision (standard deviation) listed in the table are derived from collaborative testing experiments conducted recently by EPA (Constant et al., 1974, 1975a, 1975b, 1975c). In this type of testing, a number of collaborators, following analytical procedures according to specified guidelines, sample the same ambient air spiked with known NO_2 concentrations. The collaborators are generally skilled in ambient air monitoring so that the results reported may reasonably be taken to represent an upper limit on the accuracy and precision obtainable in routine monitoring. The reader should note that the values for accuracy and precision presented in the table represent averages of data reported by all collaborators over the entire concentration ranges tested. Since the accuracy and precision obtained may depend upon tne NO_2 concentrations sampled and, moreover, in the case of the Griess-Saltzmann procedure, upon the individual collaborator performing the test, the reader is referred to the original reports for a detailed statistical discussion of the test results.

Recently, a formal audit was performed on continuous chemiluminescent analyzers (Monitor Labs 8440) in service in the St. Louis Regional Air Pollution Study (Smith and Strong, 1977) (see Chapter 8). Table 7-5 summarizes the results of the audit. Three audit levels were used: 0, 150 and 355 $\mu g/m^3$ NO_2. "Acceptable" limits for the difference between measured NO_2 values and known audit NO_2 concentrations were established for the purposes of the study at ± 19, ± 30, and ± 71 $\mu g/m^3$ respectively. At the 0 audit level all 23 analyzers were within limits; at an audit level of 150 $\mu g/m^3$, three analyzers were found to "very narrowly" exceed specified limits; at 355 $\mu g/m^3$ one different analyzer "very narrowly" exceeded specified limits. Statistics for the entire network showed average differences of less than 10 percent at both the 150 and 355 $\mu g/m^3$ audit NO_2 concentrations. Standard deviations were equal to or less than 10 percent for both non-zero audit levels.

7.2.3 Analytical Methods for NO

Numerous methods, other than the chemiluminescence procedure (discussed in Section 7.2.1), can be used for direct measurement of NO; however, none are widely used presently for air quality monitoring. These methods include: ferrous sulfate ($FeSo_4$) absorption and spectrophotometric measurement of the resulting colored complexion, (Norwitz, 1966), and ultraviolet (Sweeney et al., 1964) and infrared (Lord et al., 1974) spectroscopy. The spectroscopic techniques require long pathlengths when used to measure concentrations in typical ambient air. Mass spectrometry and gas chromatography also may be employed, but these methods are rather elaborate and expensive.

7.2.4 Sampling for NO_x

Sampling technique is a particularly important consideration for the measurement of NO and NO_2. Nitric oxide and NO_2 in the atmosphere during the day are involved in very rapid

TABLE 7-4. SUMMARY OF RELIABILITY OF NO_2 ANALYTICAL METHODS IN COMMON USE AS OBTAINED BY COLLABORATIVE TESTING (CONSTANT ET AL., 1974, 1975a, 1975b, 1975c)

Method	Range of NO_2 Concentrations Used in Test ($\mu g/m^3$)	Bias (Average for all Tests)[a]	Standard Deviation (Average for all Tests)[a]	Practical Lower Detection Limit ($\mu g/m^3$)	Comments (Reference)
Chemiluminescence (Reference Method)	80-300	$-8\ \mu g/m^3$ or -5%	14%	≤ 22	One collaborator had very large biases, and another had unstable biases. For most collaborators (8 out of 10), however, the bias was small and well balanced. (Constant et al., 1975a)
Sodium Arsenite (Equivalent Method)	50-300	$6.2\ \mu g/m^3$ or ~3%	$11\ \mu g/m^3$	≤ 9	Measurement errors were essentially uniform for all collaborators, although some dependence on NO_2 level was noted. 8 of 10 collaborators exhibited a uniform percent bias over all NO_2 levels tested. (Constant et. al., 1975c)
TGS-ANSA (Equivalent Method)	50-300	$9.5\ \mu g/m^3$ or ~ -5%	$11.6\ \mu g/m^3$	≤ 15	Errors were essentially uniform for all collaborators. The biases shown were nearly independent of NO_2 level for range tested. (Constant et al., 1974)
Griess-Saltzman (Continuous Colorimetric with dynamic calibration; both variants cited in 7.2.2.1)	90-370	$16.1\ \mu g/m^3$[b] or ~ 6%	$32.7\ \mu g/m^3$	≤ 19	Although overall results are reasonably accurate, method may produce quite inaccurate readings in an unpredictable fashion. About half of the collaborators did achieve fairly stable results. Subjectively, then, the method will produce reliable results in some hands. (Constant et al., 1975b)

[a] Depends in detail upon NO_2 concentration.

[b] Depends significantly on laboratory performing test.

TABLE 7-5. RESULTS OF 1977 FORMAL AUDIT ON IN-SERVICE CHEMILUMINESCENT ANALYZERS IN ST. LOUIS (SMITH AND STRONG, 1977)

Number Audited	Number Found Operational	Audit Level ($\mu g/m^3$)	"Acceptable" Limits for Difference ($\mu g/m^3$)	Number Outside Limits	Average Difference for Network ($\mu g/m^3$)	Standard Deviation for Network ($\mu g/m^3$)
23	23	0	±19	0	+2	6
		150	±30	3	-11	15
		355	±71	1	-28	26

reactions which keep O_3 in a photostationary state. The rate of photolysis of NO_2 (forming NO and O and thus O_3) is nearly equal to the reaction of the NO and O_3 to form NO_2. When a sample is drawn into a dark sampling line, photolysis ceases while NO continues to react with O_3 to form NO_2. Thus, long residence times in sampling lines must be avoided to obtain a representative sample. Sampling technique requirements for a given error in tolerance were discussed by Butcher and Ruff (1971). For example, Figure 7-1, adapted from these authors, shows the absolute error in NO_2 introduced in 10 seconds in a dark sampling line due to the presence of O_3 and/or NO in the line in varying concentrations. In general, due to the reactive properties of NO_x, only glass or Teflon materials should be used in sampling trains.

If NO and NO_2 are to be measured separately by a method specific for NO_2, it is necessary to remove NO_2 from the sample, then oxidize NO to NO_2 and measure the NO_2 concentration. Several selective absorbers for NO_2 have been employed, but some of the NO_2 is converted to NO in all the absorbers tested. Absorbents include Griess-Saltzman reagent (Huygen, 1970) and granules impregnated with triethanolamine (Intersociety Committee, 1977c; Levaggi et al., 1972). The triethanolamine is reported to be the best absorbent, with only two to four percent of the incoming NO_2 converted to NO.

When NO is to be measured by a method specific to NO_2, either with or without removal of NO_2 from the sample, it is necessary to oxidize NO to NO_2 in the gas phase. The most frequently used oxidizer is chromic oxide on a fire brick granule support (Intersociety Committee, 1977c; Levaggi et al., 1974). This material gives over 99 percent oxidation when the relative humidity in the sample is between 20 and 80 percent. The chromic oxide also removes SO_2.

Considerations relating to the reduction of NO_2 to NO have been discussed in Section 7.2.1.

7.2.5 Calibration of NO and NO_2 Monitoring Instruments

Calibration of monitoring instruments or methods may be accomplished either by measuring a gas of known concentration or by comparing measurements of a stable source with measurements of the same source made by a primary reference method.

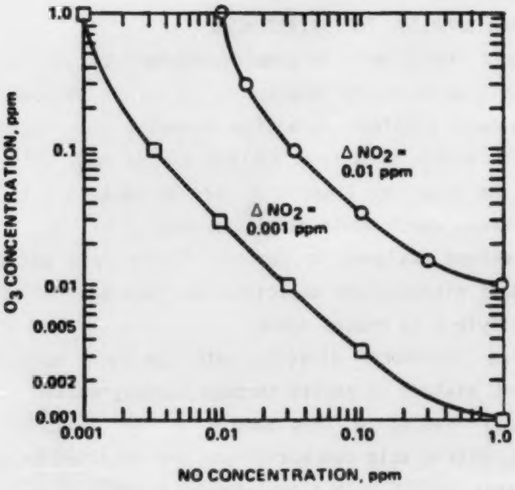

Figure 7-1. Absolute error in NO_2 and ΔNO_2 for 10 seconds in dark sampling line (Batcher and Ruff, 1971).

Standard gas sources are the principal means by which NO and NO_2 measurement instruments are calibrated. The preparation of standard mixtures of NO in nitrogen was studied by the National Bureau of Standards (Hughes, 1975). The initial accuracy with which standards may be prepared, based on either pressure or mass measurements, is quite good. The stability of mixtures at concentrations above about 50 ppm is satisfactory with only 0 to 1 percent average change in concentrations over a seven-month period. Other sources which have been employed occasionally include permeation of compressed NO through membranes to produce dilute NO streams (Hughes, 1975), electrolytic generation (Hersch and Deuringer, 1963), catalytic reduction of a known concentration of NO_2 (Breitenbach and Shelef, 1973), or photolysis of known NO_2 concentrations and rapid dilution (Guicherit, 1972).

The permeation tube is the only direct source of dilute NO_2 mixtures in widespread use (O'Keefe and Ortman, 1966; Scaringelli et al., 1970; Shy, 1970; Shy et al., 1970). It may be calibrated by weighing or by rarely used micromanometric measurements. The other common procedure used to calibrate NO_2 measurement instruments is gas-phase titration. Stable sources of known concentrations of both NO and O_3 are required. A dilute stream of NO is measured by NO methods. The O_3 is added to the stream at a constant rate. The decrease in NO by reaction with the added ozone is equal to the NO_2 formed. Thus, a known NO_2 concentration is created. The U.S. EPA (1976a) recommends the combined use of permeation tubes and gas-phase titration, using one technique to check the other.

7.3 ANALYTICAL METHODS AND SAMPLING FOR NITRIC ACID

Monitoring for ambient nitric acid is complicated both by the low concentrations present and by the fact that nitric acid in the atmosphere is in the gaseous state. Collection of a representative sample without artifact formation presents some technical difficulties. In general, also, sampling of nitric acid from ambient air is made difficult by its tendency to adhere to the walls of the sampling lines. It may be necessary to heat sampling lines to prevent condensation of water, which would result in removal of HNO_3.

A microcoulometric method designed to measure nitric acid was developed by Miller and Spicer (1975, 1976). A Mast microcoulomb detection cell was adapted for sensing acid gases in samples pretreated with ethylene to remove ozone.

Readings from samples introduced directly into the cell indicate total acid content. Another sample of the test mixture is passed through loosely-packed nylon fiber which removes the nitric acid. The cell reading of this sample is representative of total acid content except nitric acid. Thus, nitric acid concentrations are obtained by subtraction.

The sensitivity of this method at a signal-to-noise ratio of two to one is approximately 2 ppb. No detectable interferences have been reported from SO_2, NO_2, PAN, H_2SO_4, and formaldehyde (CH_2O). It should, however, be noted that Spicer et al. (1978a) report significant artifact nitrate formation on nylon filters under conditions of very high NO_2 concentrations (56,400 $\mu g/m^3$; 30 ppm) and high humidity. Although concentrations of this magnitude would not be expected to occur in ambient situations, the possible implications of these reported interferences for measurements obtained by the microcoulometric method have yet to be evaluated.

Using air streams passing through a cellulose filter impregnated with sodium chloride, Okita et al. (1976) report collection efficiencies for nitric acid ranging from 93 to 100 percent. Interferences from NO_2 were reported over a range of NO_2 concentrations up to 15,000 $\mu g/m^3$ (8.0 ppm). The equivalent of about 1 μg NO_3-N/m^3 of artifact gaseous nitrate corresponded to 188 $\mu g/m^3$ (0.1 ppm) NO_2 being passed through the filter at relative humidities of 55 to 72 percent. Ozone did not enhance artifact formation at a concentra tion of 980 $\mu g/m^3$ (0.73 ppm) in the presence of 1,372 $\mu g/m^3$ (0.73 ppm) NO_2. Interferences from PAN and n-propyl nitrate were cited as negligible or very small. Nitrate formation from NO_2 increased with increasing relative humidity.

Recently, Tuazon et al. (1978) have reported measurements of nitric acid under ambient conditions with a detection limit of 2 ppb. The system achieves this sensitivity by means of a folded-path optical system which results in pathlengths of up to 2 km in the sample cell. Fringes produced in a high-resolution infrared Michelson interferometer, coupled to the sample cell, are scanned optically. The resultant variations in signal are related to the Fourier transform of the spectrum which is recovered automatically by appropriate data processing.

A summary of the current status of methods to measure atmospheric nitric acid has been published very recently (Stevens and McClenny, 1979). Table 7-6 summarizes the techniques considered.

Methodology for measuring nitric acid is still in the development stage. Studies conducted to date are not sufficient to assess with any degree of confidence the suitability of the methods described in this section for routine ambient monitoring.

7.4 ANALYTICAL METHODS AND SAMPLING FOR NITRATE

Nitrate analyses have been performed routinely for many years and a large number of chemical methods have been reported. Since analytical methods for inorganic nitrate generally proceed by aqueous extraction, the final chemical quantitative determination of ion concentrations is similar for samples drawn from air, water, and soil.

7.4.1 Sampling for Nitrate From Airborne Particulate Matter

Particulate nitrate as a fraction of total suspended particulates has been sampled in this country largely by standardized sampling techniques using high volume (HIVOL) samplers. USEPA minimum specifications for the HIVOL are well documented (Federal Register, 1971). A continuous 24-hour sample of ambient air, typically at flow rates of approximately 1 to 2 standard cubic meters per minute, is drawn through a glass fiber filter which traps the particulate matter. The upper size limit of particulate matter collected depends on the geometry of the sampler housing but is generally above about 30 μm in aerodynamic diameter, well above the respirable size range. The sampler thus collects all of the respirable material and some

TABLE 7-6. SUMMARY OF NITRIC ACID DETECTION TECHNIQUES[a]

Technique	Procedure	Minimum Detectable Level (ppbv)	Interferences Tested For, To Date	Reference
Chemiluminescence	Simple modification of chemiluminescence procedure used for NO	5.0	NO, NO_2, PAN, organic nitrates	Joseph and Spicer, 1978
Chemiluminescence	Adaptation of sensitive chemiluminescence NO_x monitor described by Ritter et al., 1978	0.3	NO, NO_2, PAN, organic nitrates	Kelly and Stedman, 1979 Likens, 1976
Fourier Transform Spectrometer	Long path infrared spectrometry	5.0	Most gaseous species in normal ambient air	Tuazon et al., 1979, 1978
Microcoulometry	Sample conditioning with ethylene to remove ozone interference; difference method using direct reading and reading after nylon trap which removes HNO_3	5.0	O_3, NO_2, SO_2, H_2SO_4, HCl, HCHO, PAN, HCOOH, HNO_2	Miller and Spicer, 1975 Spicer et al., 1978a, 1978b
Colorimetry	Reduction to NH_4^+ of fixed organic nitrogen collected on nylon filter, followed by indophenol ammonia test. Teflon prefilter.	<0.1	NH_4^+, particulate nitrate	Lazrus et al., 1968, 1979
Colorimetry	Collection of HNO_3 on NaCl impregnated filters, followed by extraction and hydrazine reduction-diazotization analysis of nitrate	0.1	NO_2, particulate nitrate	Okita et al., 1976[70] Forrest et al., 1979
Electron Capture Gas Chromatography	Collection of HNO_3 on nylon or cotton, extraction, conversion to nitrobenzene analysis by gas chromatography	0.1	NO_2, particulate nitrate	Hare et al., 1979 Tesch and Sievers, 1979 Ross et al., 1975

[a]Adapted from Stevens and McClenny, 1979

fraction of the non-respirable material suspended in ambient air. In typical nitrate monitoring, a portion of the HIVOL filter is subjected to aqueous extraction and the water-soluble nitrate analyzed as described below in Section 7.4.2.

Recent reports point to serious difficulties associated with the routine use of glass fiber filters. In a study of nitrate in auto exhaust, Pierson et al. (1974) report that glass fiber filters collected about twice the amount of nitrate when compared to quartz fiber filters. Nitrate also was found on glass fiber filters which were inserted downstream of either quartz or glass fiber primary filters, providing additional evidence of artifact formation from gaseous constituents. Spicer (1976) reported that glass fiber filters completely removed gaseous nitric acid when in low concentration in gas streams, while Teflon and quartz filters showed no corresponding effect. O'Brien et al. (1974) describe very unusual results of particle size distribution determinations of photochemical aerosol collected in the Los Angeles basin using a cascade impactor where all particle size fractions were collected on glass fiber filters. The authors speculated that conversion of gaseous nitrate precursors on the filter masked the true nitrate size distribution.

Okita et al. (1976) report that untreated glass fiber filters collect nitric acid vapor with a highly variable collection efficiency (0-56 percent), suggesting erratic nitrate artifact formation in urban atmospheres containing nitric acid.

In an intensive laboratory investigation of interferences in atmospheric particulate nitrate sampling, Spicer, Schumacher and co-workers (1976, 1978a) concluded that all five types of glass filters investigated exhibited serious artifact formation due to collection of gaseous nitric acid and, to some extent, NO_2 as nitrate. Cellulose acetate and nylon filters were also reported to exhibit severe interferences from nitric acid. Negligible interferences were reported for polycarbonate and Teflon filters. Interferences on quartz fiber filters varied with the filter type, with ADL Microquartz showing the least effect at NO_2 concentrations of 592 $\mu g/m^3$ (0.315 ppm). When a variety of quartz filter types were tested, the greatest quantity of artifact nitrate was formed on the Gelman AE filter. Artifact nitrate formed on this filter was calculated to be less than 2 $\mu g/m^3$ (0.001 ppm) during a standard 24-hour HIVOL measurement. The estimate was derived from drawing air samples of about 1 m^3 containing 4,512 $\mu g/m^3$ (2.4 ppm) NO_2 through the filters. The relative humidity was 30 ± 10 percent.

Most recently, Spicer and Schumacher (in press) reported the results of a comparison of nitrate concentrations in samples collected on various filter types in Upland, California during October and November, 1976 (Table 7-7). During the experiment, meteorological conditions varied from warm, hazy weather to hot, dry, very clean desert wind conditions. Nitrate analyses were performed by ion exchange chromatography. All filter types used had comparable particle collection efficiencies according to the manufacturer's specifications. The ratio of nitrate collected on Glass 1 to that collected simultaneously with identical HIVOL samplers on Quartz 2 ranged from 4.8 to 36.6 and averaged 18.9. The ratio of nitrate collected on Glass 2 to Quartz 2 ranged from 2.8 to 49 and averaged 10.9.

TABLE 7-7. COMPARISON OF NITRATE COLLECTED ON VARIOUS FILTER[a] TYPES (SPICER AND SCHUMACHER, IN PRESS; SPICER ET AL., 1978a)

Date	Filter	NO_3^- μg/m³	Date	Filter	NO_3^- μg/m³	Date	Filter	NO_3^- μg/m³
Oct. 15	Quartz 1 Quartz 2	1.6 1.6	Oct. 26	Quartz 1 Quartz 2	1.3 2.1	Nov. 4	Glass 2 Quartz 2	3.0 1.1
Oct. 18	Glass 1 Quartz 2	14.4 0.39	Oct. 27	Glass 2 Quartz 2	3.9 0.52	Nov. 5	Glass 2 Quartz 2	6.1 0.98
Oct. 19	Glass 1 Quartz 2	17.0 1.2	Oct. 28	Glass 1 Quartz 2	9.1 1.9	Nov. 9	Glass 2 Quartz 2	18.4 3.1
Oct. 20	Glass 1 Quartz 2	28.7 2.3	Oct. 29	Quartz 1 Quartz 2	1.8 2.9	Nov. 10	Quartz 1 Quartz 2	1.9 2.3
Oct. 21	Glass 1 Quartz 2	18.8 0.82	Nov. 1	Quartz 2 Quartz 2	1.7 1.7	Nov. 11	Quartz 2 Quartz 2	2.0 1.9
Oct. 22	Glass 1 Quartz 2	11.2 0.49	Nov. 2	Glass 2 Quartz 2	9.1 1.6	Nov. 12	Glass 2 Quartz 2	6.0 1.3
Oct. 25	Glass 2 Quartz 2	38.4 0.78	Nov. 3	Quartz 1 Quartz 2	0.68 1.1	Nov. 15	Glass 2 Quartz 2	14.3 3.0

[a]Glass 1 - "EPA Type" Gelman AA.

Glass 2 - Gelman A.

Quartz 1 - High purity quartz filter developed by Arthur D. Little under contract to EPA.

Quartz 2 - Pallflex QAST.

Recently, Harker et al. (1977) have reported laboratory observations of loss of particulate nitrate from collecting filters through chemical reaction with sulfuric acid aerosol, formed from the photochemical oxidation of SO_2. Most recently, Appel and coworkers (1979, in press) have conducted several studies bearing on both positive artifact formation and loss of nitrate from a variety of filter media (Appel et al., 1979, Appel et al., in press). They concluded that gaseous HNO_3 is the principal source of artifact nitrate formation, NO_2 collection only became substantial at high ozone levels. Ambient particulate nitrate values (at San José and Los Alamitos, California) differed by up to a factor of 2.4 depending upon filter medium and sampling rate, in contrast to the much larger sampling errors reported by Spicer and Schumacher (in press). Fluoropore (Teflon) filters in low volume samplers were subject to small error although, under laboratory conditions, passage of NH_3- and HNO_3-free air through the filter could result in the loss of up to 50 percent of the particulate nitrate. This is consistent with the relatively high vapor pressure of ammonium nitrate (see page 6-49). They also reported that at low HNO_3 levels nitrate, on glass fiber filters, indicated (within about 3 percent) total nitrate, i.e., particulate nitrate plus HNO_3 rather than particulate nitrate alone. They concluded that the degree of error associated with glass fiber filter media could be expected to vary with location, time of year and day, paralleling changes in nitric acid levels.

These results point to the conclusion that most of the existing data on urban ambient nitrate concentrations must be considered to be of doubtful validity. It is, furthermore, doubtful that any of these data can be corrected even if mechanisms for artifact formation are clarified in the future since nitric acid, which presently appears to play a significant role in positive artifact formation, is not routinely monitored.

It is, however, possible that data from certain monitoring sites may be validated in special cases where it can be shown that the species responsible for the artifact processes were all sufficiently low during the monitoring period of interest.

7.4.2 Analysis of Nitrate from Airborne Particulate Matter

Although most of the nitrate analysis methods described in this section found application originally in analyzing nitrate in samples of natural waters, they have also been used to analyze nitrate in aqueous extracts of particulate matter (Hermance et al., 1971) and in solutions obtained through absorption of nitrogen oxides contained in streams of air (Kieselbach, 1944b; Kothny, 1974).

The oldest procedures for analyzing nitrate used brucine ($C_{23}H_{26}N_2O_4$) (Intersociety Committee, 1977a, Jenkins and Medsker, 1964; Lunge and Lwoff, 1894; Robinson et al., 1969) or phenoldisulfonic acid (ASTM, 1968; Beatty et al., 1943; Easthoe and Pollard, 1950; Intersociety Committee, 1977d; Taras, 1950). Newer procedures extensively used to analyze nitrate in atmospheric particulate matter extracts involve the nitration of xylenols [$(CH_3)_2C_6H_3OH$] and separation of the nitro-derivative by extraction or distillation (Andrews, 1964; Buckett et al., 1955; Hartley and Asai, 1960; Holler and Huch, 1949; Intersociety Committee, 1977b; Swain, 1957; Yagoda and Goldman, 1943). Recent comparison of a 2,4-xylenol procedure (Intersociety Committee 1977b) with the automated copper-cadmium reduction and diazotization method (Technicon, 1973) in samples collected near high density vehicular traffic, demonstrated a negative interference in the former up to a factor of 3 (Appel et al., 1977).

Nitration of chromotropic acid [$C_{10}H_4(OH)_2(SO_3H)_2$] (West and Ramachandran, 1966) and coumarin ($C_9H_6O_2$) analogs (Laby and Morton, 1966; Skujins, 1964) also have been reported. Small amounts of nitrate can be assayed by the quenching of the fluorescence after nitration of fluorescein ($C_{20}H_{12}O_5$) (Axelrod et al., 1970). Nitrate analysis can also be accomplished through reduction with Devarda alloy to ammonia (NH_3) (Kieselbach, 1944a; Richardson, 1938) or reduction of nitrate to nitrite by zinc (Chow and Johnstone, 1962), cadmium (Morris and Riley, 1963; Strickland and Parsons, 1972; Wood et al., 1967) or hydrazine (NH_2NH_2) (Mullin and Riley, 1955). Automation instituted by the NASN improved the hydrazine reduction process by curtailing the unwanted effects resulting from its sensitivity to motion (Morgan et al., 1967b). The addition of antimony sulfate [$Sb_2(SO_4)_3$] eliminates the chloride interferences found in most nitration methods (West and Ramachandran, 1966). One brucine procedure circumvents the effect of chlorides by adding an excess of sodium chloride before nitration (Intersociety Committee, 1977a).

Nitrate analysis by ion-selective electrodes has been used but has several disadvantages: potential drifts caused by agitation speed, necessity of frequent re-standardization, interferences caused by nonspecificity of electrodes which respond to other ions in the aqueous extracts, and non-stoichiometric absorption of the gases in the collecting reagent (DiMartini, 1970; Driscoll et al., 1972; Gordievski et al., 1972). In atmospheric analysis, the electrode procedure has no advantage over direct UV determination of either nitrite formed in an alkaline absorbent (Altshuller and Wartburg, 1960) or nitrate (Cawse, 1967) obtained after oxidation and absorption of nitrogen oxides in alkaline permanganate (Kieselbach, 1944b; Kothny, 1974). Microscopic techniques also allow analysis of individual nitrate particles (Bigg et al., 1974).

Small et al (1975) report an application of ion exchange chromatography to the measurement of a wide variety of cations and anions including the nitrate and nitrite ion. The novel feature of the method is the use of a second ion exchange "stripper" column (after a conventional separating column) which effectively eliminates or neutralizes the eluting ions. Since only the species of interest in a background of deionized water leaves the stripper column, concentration determinations may be made by a simple and sensitive conductometric technique. Mulik et al (1976) report the application of this technique to measurement of water-soluble nitrate on HIVOL filters. The separator column, containing a strong basic resin, separates anions in a background of carbonate eluant. The stripper column, containing a strong acid resin, converts the sample ion and the carbonate eluant to nitric and carbonic acid, respectively. Since carbonic acid has low conductivity, the nitrate ion alone is effectively measured in a conductivity detector. Under the experimental conditions, sensitivity of 0.1

μg/mℓ was reported. The related standard deviation was 1 percent (95 percent confidence level) for ten replicate injections at the 5 μg/mℓ level. At this concentration level, no interferences were found from fluoride, chloride, nitrite, sulfite, sulfate, silicate, or carbonate. Positive interferences were found for bromide and phosphate but the authors suggest techniques for eliminating these.

In other recent work, Glover and Hoffsommer (1974) and Ross et al (1975) report a technique for assay of aqueous nitrate and nitrite extractions by conversion to nitrobenzene. Both techniques involve the nitration of benzene in the presence of sulfuric acid to form nitrobenzene, a relatively stable compound, followed by gas chromatographic analysis. Careful calibration is required in both procedures, since a significant fraction of the nitrobenzene formed may be lost to the acid layer. Ross et al. recommend a calibration procedure whereby a standard is subject to the same reaction procedures as the unknown, while Glover and Hoffsommer use internal calibration with added nitrotoluene. The lower detection limits reported by Ross et al. are in the range of 10^{-12}g nitrobenzene in a 1 μℓ sample. Conversion efficiences for KNO_3, KNO_2 and HNO_3 were reported as 90.3 ± 7.9, 100.4 ± 4.2 and 99.9 ± 5.2 percent, respectively. Glover and Hoffsommer report similar recovery rates for KNO_3 and KNO_2.

Several methods in current use for analysis of nitrate in water and soil are applicable also to analysis of nitrate derived from ambient air samples (Sections 7.4.3 and 7.4.4).

7.4.3 Nitrate in Water

Current methodology for determination of nitrate in water is summarized in Table 7-8. No single method is satisfactory over the broad range of concentrations and water matrices to be found in environmental samples. Since nitrate species are highly labile, a variety of techniques have been used to preserve them during storage, including refrigeration, freezing, and addition of sulfuric acid and of mercuric chloride. Simple refrigeration is adequate for periods up to a day; freezing is effective for longer preservation (Brezonik and Lee, 1966). Mercuric chloride is effective, especially when coupled with refrigeration or freezing, but the mercuric ion slowly degrades columns used in various reduction methods (Table 7-8) and also is toxic (Howe and Holley, 1969; U.S. EPA, 1974).

The strong absorption of the nitrate ion in the range 210-220 nm allows direct spectrophotometric measurement (Altshuller and Wartburg, 1960; Bastian et al., 1957; Mertens and Massart, 1973). Iron and nitrite are significant interferants. Absorbance of nitrate is also influenced by changes in acidity. At 210 nm, a variation of 20 percent in perchloric acid concentration causes an error of approximately 5 parts per thousand in nitrate measurement. One author's report indicates that the spectrophotometric method is three times more sensitive than the brucine method (Noll, 1945).

Nitrate ion selective electrodes have also been used for measurement of nitrate in water (Kenney et al., 1970; Langmuir and Jacobson, 1970). The method, however, is not currently in widespread use.

TABLE 7-8. ANALYTICAL METHODS FOR NITRATE IN WATER

Method	Range (mg/ℓ nitrate-nitrogen)	Interferences	Reference
1. UV absorbance	0.1 - 10	Organic matter	APHA, 1976
2. Ion selective electrode	0.2 - 1400	Chloride, ionic strength	APHA, 1976
Nitration and Oxidation Reactions			
3. Phenoldisulfonic acid		Chloride	APHA, 1976
4. Brucine	0.1 - 2.0		APHA, 1976
5. Chromotropic	0.1 - 5.0	Many, but all readily removed	APHA, 1976
6. Automated fluorimetric with substituted benzophenone	0.05- 4.0	Organic color, chloride, sulfide, but readily removed	Afghan and Ryan, 1975
7. Szechrome	0.05- 1.0	Unknown	Szekely, 1975
Reduction Methods			
8. Zinc	0.02- 1.0	Sensitivity varies with age of column	O'Brien and Fiore, 1962
9. Amalgamated cadmium	<0.01- 1.0	Sensitivity varies with age of column	APHA, 1976 Technicon, 1973
10. Copperized cadmium	<0.01- 1.0	Sensitivity varies with age of column	EPA, 1974 APHA, 1976 Strickland and Parsons, 1972
11. Hydrazine-copper	<0.01- 1.0	Reduction is pH sensitive	Kemphake, 1976
12. Devarda's alloy	2 ->200		APHA, 1976

Wet chemical methods for nitrate analysis are colorimetric and fall generally into two major reaction categories: (1) nitration reactions which involve the substitution of a hydrogen in an aromatic compound by the NO_2 moiety, and (2) reduction of nitrate to nitrite.

Nitration and oxidation reactions generally require a strong acid medium as well as heating. Since it is desirable that only one extraction be used for all forms of mineral nitrogen, methods of analysis not subject to chloride interference are preferred because many air samples may contain chloride derived from suspended sea salts. Some nitration methods are subject to serious chloride interferences. While chloride interferences can be eliminated from the brucine method (Intersociety Committee, 1977a), experience has shown that results obtained using this method are difficult to reproduce. Interferences from nitrite and chlorine can be eliminated in the chromotropic acid method (APHA, 1976).

Procedures which involve reduction of nitrate to nitrite are widely used because there is a simple, sensitive and well-tested analytical procedure for determination of the nitrite (NO_2^-) ion (Section 7.2.1). Although nitrate is readily reduced by a variety of agents including hydrazine, metallic zinc or cadmium, difficulties with quantitative recovery have been reported (Henriksen, 1965; Mullin and Riley, 1955; Nydahl, 1976; O'Brien and Fiore, 1962; Strickland and Parsons, 1972; Technicon, 1973; U.S. EPA, 1974). Techniques presently recommended to avoid these difficulties have been documented by the American Public Health Association (Stainton, 1974). Columns using copperized cadmium (Strickland and Parsons, 1972; U.S. EPA, 1974) or amalgamated cadmium granules (National Research Council, 1978) or copperized cadmium wire (Bremner 1965) give stoichiometric or near stoichiometric reductions. These methods are widely used and are regarded as accurate, sensitive and acceptable procedures (APHA, 1976; National Research Council, 1978; U.S. EPA, 1974). Nitrate can be reduced quantitatively to ammonia by Devarda's alloy and subsequently analyzed by titration or colorimetrically.

7.4.4 Nitrate in Soil

As in the case of certain techniques for measurement of nitrate in water, some techniques used in analysis for nitrate in soil can be adapted for atmospheric work.

Nitrate levels in soil samples can change rapidly through nitrification, denitrification and flushing of nitrate. Biocides have been used to prevent microbial activity but they are often ineffective (Bremner, 1965). Cold storage (Allen and Grimshaw, 1962) and rapid air or oven drying have also been used. Most extraction methods employ a salt solution such as $CaSO_4$, K_2SO_4 or KCl (Bremner, 1965). Methods of removing turbidity include flocculation with aluminum hydroxide (Cawse, 1967) or calcium salts as well as the use of activated charcoal, ion exchange resins or hydrogen peroxide, but the last three techniques may cause changes in nitrate content (Bremner, 1965). Ultraviolet methods used in analysis of atmospheric nitrate samples require similar flocculation techniques to remove turbidity, color and other interferences.

Analytical methods are summarized in Table 7-9.

TABLE 7-9. METHODS FOR DETERMINATION OF NITRATE IN SOILS

Method	Range, mg/ℓ[a]	Interferences	References
1. Ion electrode	2 -1400	Chloride, bromide, nitrite, iodide, sulfide, ionic strength	Carlson and Keeney, 1971
2. Phenoldisulfonic acid	0.1 - 2	Chloride, organic matter, nitrite	Bremner, 1965
3. Brucine	0.1 - 2		Bremner, 1965; Bremner and Keeney, 1965, APHA, 1976
4. Reduction of NO_2^- by Cd Griess-Illosvay method	0.02-0.1	None	Bremner, 1965; Bremner and Keeney, 1965 APHA, 1976
5. Reduction to NH_3 by Devarda alloy, steam distillation of NH_3	1 -1000	Labile amide, phosphate, nitrite	Bremner, 1965
6. UV absorbance			Cawse, 1967

[a] Range on soil basis varies widely depending on soil:solution ratio of extractant.

7.4.5 Nitrate in Plant and Animal Tissue

Methods analogous to those described above have been applied to measurement of nitrate in plant and animal tissue. Nitrate ion concentrations have been measured in tobacco extracts using a spectrophotometric procedure (Barkemeyer, 1966). Wegner (1972) has described a procedure for determining both nitrate and nitrite in biological fluids. Fudge and Truman (1973) described the analysis of nitrate and nitrite in meat products. Methods of analysis for plant tissue have been recently described by Carlson and Keeney (1971).

7.5 SAMPLING AND ANALYTICAL METHODS FOR NITROSAMINES

The sampling and analytical techniques for nitrosamines depend on the medium in which the nitrosamine to be sampled and analyzed is found.

Since it is of some importance to place human exposure via the atmospheric medium in perspective with regard to other media such as food and potable water, the discussion of analytical techniques in this section includes methodology appropriate to the three most important media: air, food and water.

7.5.1 Nitrosamines in Air

Because of the low nitrosamine concentrations in air, sample concentration methods are necessary. One suitable concentration procedure is the adsorption of nitrosamines on a solid substance. Bretschneider and Matz (1976) report using chemically pure active carbon. Another effective technique is the use of chemically bonded stationary phases (Pellizzari et al., 1975). In this method, nitrosamines are collected by passing air through a cartridge packed with a solidsorbent. Samples are desorbed by flash heating the cartridge contents into a gas chromatographic column. The chromatograph may then be interfaced to a mass spectrometer for component identification and measurement. The sorbents Tenax and Chromosorb have been evaluated at sampling rates up to 9 ℓ/min. Results showed that they maintained efficiencies of > 90 percent. Carbowax 600 and 400, and oxypropionitrile, coated or chemically bonded to a support, were also highly efficient (> 90 percent).

EPA's National Enforcement Investigations Center (1977a, 1977b) reports the use of a basic collecting medium (1N KOH) shielded by foil to preclude irradiation by light. The KOH solutions are subsequently extracted with dichloromethane. Before evaporative concentration, 2,2,4 trimethylpentate was added as a keeper. It is important in using either solid sorbents or liquid KOH traps, that the procedure be carefully checked to ensure that the measurement is free of artifacts.

Fine et al. (1974) have developed a method for detecting N-nitroso compounds based on catalytic cleavage of the N-NO bond and the subsequent infrared detection of chemiluminescence. The technique, called thermal energy analysis (TEA), is coupled with gas chromatography and, sometimes, with high-pressure liquid chromatography (Fine and Rounbehler, 1975). The technique is highly sensitive with a detection limit of about 1 ng/mℓ. The TEA detector operates by splitting the nitrosyl radical off N-nitroso compounds coming from a chromatographic column.

The nitrosyl radical is then reacted with ozone, yielding excited nitrogen dioxide which subsequently decays to the ground state by emission of near-infrared radiation. The intensity of this radiation is proportional to the number of nitrosyl radicals present. Artifact formation has not been reported to be a significant problem in the method (Pellizzari, 1977). TEA responses have also been reported for some compounds containing the O-nitroso, O-nitro, C-nitroso, and N-nitroso groups and two recent papers describe approaches for distinguishing N-nitroso responses (Hansen et al., 1979; Krull et al., 1979).

7.5.2 Nitrosamines in Water

The usual precautions employed in the collection of samples for organic analysis should be followed when sampling water for nitrosamine analysis. Stabilization to pH 8 may be needed and samples should be protected from light and kept cold because of the photosensitivity of nitrosamines.

Several analytical procedures for the determination of N-nitroso compounds in water have been reported. Fine et al (1975) reported two concentration and extraction procedures--one based on a liquid-liquid extraction, and the other based on the adsorption of the organic fraction and its subsequent extraction with chloroform. Gas chromatography and high-pressure liquid chromatography, each combined with detection by TEA, have been used by Fine and co-workers (1975) to measure part per trillion concentrations of volatile and non-volatile non-ionic nitroso compounds, respectively, in water supplies.

Older techniques for the detection and estimation of N-nitrosamines in water are polarography (Walter, 1971) and colorimetry (Mohler and Mayrhofer, 1968), but neither method has the sensitivity required for environmental samples. Furthermore, the colorimetric method has exacting experimental conditions and cannot be used for complicated mixtures.

7.5.3 Nitrosamines in Food

Determination of nitrosamines in foodstuffs is made difficult by the complexity of food, many components of which contain nitrogen and react chemically in a manner similar to nitrosamines. Many methods have been used to detect nitrosamines in food, including polarography, UV absorption, thin layer chromatography, and gas chromatography, but these methods generally have been plagued with contamination and artifact problems. Gas chromatography-mass spectrometry (GC-MS) is presently the most acceptable procedure for the measurement of nitrosamines in food. Wassermann (1972) and Eisenbrand (1975) have published surveys of analytical techniques used in the isolation and detection of nitrosamines.

GC-MS also appears to be the most acceptable method for analysis of nitrosamines in tobacco smoke, but nitrogen-specific GC detectors have also been used (Spincer and Westcott, 1976).

7.6 SUMMARY

Since the publication in 1971 of the original document Air Quality Criteria for Nitrogen Oxides, there have been significant changes in the technology associated with measurement of ambient concentrations of NO_x and NO_x-derived pollutants.

With regard to the measurement of NO_2, the original Reference Method, the Jacobs-Hochheiser technique, was discovered to have unresolvable technical difficulties and was withdrawn by the U.S. Environmental Protection Agency on June 8, 1973. Since that time, adequate methodology has been validated for measuring both NO and NO_2 in concentrations encountered in ambient air. Accurate techniques utilizing standardized gas sources, permeation tubes, or a gas-phase titration have commonly been used for calibration. The chemiluminescence technique is specific for NO. Nitrogen dioxide concentrations can be determined also with appropriate modifications of this method. Such a modification was adopted on December 1, 1976, as the Reference Method for NO_2 measurements. As of December 14, 1977, the sodium-arsenite procedure and the TGS-ANSA method were designated equivalent methods, suitable for 24-hour instrumental averaging times. The chemiluminescence method must be used with care when modified for measurement of NO_2 since a number of compounds which may be present in the atmosphere may interfere with the instrument's accuracy. Variations of the Griess-Saltzman method are specific for NO_2. Under certain circumstances, ozone can be a significant negative interferent in the method. Dynamic calibration of the Griess-Saltzman methods in current use is considered necessary for reliable measurement.

Although adequate chemical techniques exist for the determination of the nitrate fraction of suspended particulate matter in ambient air, a number of very recent reports have pointed to significant positive nitrate artifact formation on the glass fiber filters in widespread use for collecting the particulate. In addition, negative artifacts result from the volatilization of the ammonium nitrate. At this time, therefore, almost the entire urban data base on ambient nitrate concentrations must be considered to be of doubtful validity.

Recent discovery of N-nitroso compounds in food, water, tobacco products, and ambient air has prompted the development of a variety of new instrumental techniques in the last few years. Measurement technology is still developing and insufficient time has elapsed for careful evaluation of existing techniques, particularly in the area of sampling.

Development of long pathlength infrared absorption techniques has recently made possible the observation of nitric acid in ambient air, but the procedure is expensive and does not currently lend itself to routine atmospheric measurement. Other analytical methods are available for routine monitoring but have yet to be carefully evaluated.

CHAPTER 7

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8. OBSERVED ATMOSPHERIC CONCENTRATIONS OF NO_x AND OTHER NITROGENOUS COMPOUNDS

8.1 ATMOSPHERIC CONCENTRATIONS OF NO_x

In this section, selected examples of ambient concentrations of NO_x are presented in order to place possible human exposure in nationwide perspective. NO_2 is given particular emphasis since it is the oxide of nitrogen of most concern to human health. The data presented are not intended to be a compendium of ambient monitoring activities. They have been summarized to give a representative picture of NO_2 concentrations in the United States and, in particular, to provide a rational basis for deciding whether or not existing ambient NO_2 levels are a cause for concern when viewed in the context of the health and welfare effects reviewed elsewhere in this document.

The relationship, nationwide, between short-term peaks and annual average concentrations is reviewed to estimate the variability in peak-to-mean ratios of NO_2 concentrations. If the variability is small, then a rational basis for protecting human health and welfare might be derived from either considerations of the lowest peak concentrations or of the lowest annual means considered tolerable. If the variability is large, then this fact would point to the necessity of considering separately the effects of peak and long-term NO_2 concentrations.

The question of whether or not any "typical" diurnal pattern of NO_2 concentrations exists nationwide is discussed. This question has bearing on the problem of estimating human exposure to ambient NO_2 concentrations. Other considerations bearing on human exposure are also illustrated with recent data on the temporal and spatial variability of NO_2 concentrations in a single airshed. Data are also presented to illustrate certain general types of atmospheric mechanisms potentially leading to high short-term NO_2 concentrations. An example is given of late morning NO_2 peaks which typify photochemical pollution processes such as occur frequently in the Los Angeles area and elsewhere. Also illustrated is another, less familiar, mechanism, ozone titration, in which NO is rapidly oxidized to NO_2 by ambient ozone. This mechanism is of interest because of its potential for producing high NO_2 concentrations both in urban areas and in plumes from point sources. Peak concentrations so produced generally occur later in the day than those produced by photochemical processes.

Where examples of atmospheric mechanisms leading to high NO_2 concentrations are described, the examples are to be viewed as illustrative only. Detailed evaluations of the relative importance of different mechanisms as well as the relative impacts of various source types and appropriate ameliorative actions are outside the scope of this document and are best considered in the State Implementation Plans on a case-by-case, area-by-area basis.

It is important to note that although a careful attempt has been made to include only high quality monitoring data in this chapter, the data cited have not usually been derived from monitoring activities subject to a formal quality assurance program. Identification of the analytic procedures used to obtain the data cited in this section is generally available

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in the tables accompanying the text. Reference is made to Chapter 7 of this document for a discussion of the reliability of these procedures. In the absence of a formal program of audit for the wide variety of laboratories conducting ambient monitoring, the estimates of reliability given in Chapter 7 are best considered as upper bounds. Specifically, the California Air Resources Board's (CARB) Air Monitoring Technical Advisory Committee (AMTAC) composed of the CARB, the Air Industrial Hygiene Laboratory, EPA Region IX, and the local air pollution control districts, have reported NO_x, NO, and NO_2 concentrations to be on the average biased about 14 percent higher than the true value. The bias was independently determined by three agencies participating in AMTAC and applies to any analyzer calibrated by the manual Saltzman procedure. This affects virtually all California sites. A joint report is being prepared by the Air Industrial Hygiene Laboratory Section of the Department of Health Services and the CARB regarding factors affecting the Saltzman reagent. The formulation of the reagent used in the calibration procedure contained 0.5 percent sulfanilic acid, 5.0 percent acetic acid, and 0.005 percent NEDA (see Chapter 7). The bias reported applies to both static and dynamic calibration procedures. All the California data cited in this chapter are to be considered subject to biases of the reported magnitude.

8.1.1 Background Concentrations of NO_x

Data on background concentrations of nitrogen oxides are extremely limited. Robinson and Robbins (1972) summarized measurements of NO and NO_2 concentrations from various locations, such as Panama, the mid-Pacific, Florida, Hawaii, Ireland, North Carolina, Pike's Peak, and Antarctica. From these data, they estimated that the mean background NO and NO_2 concentrations for land areas between 65^oN and 65^oS are 3.8 $\mu g/m^3$ (0.002 ppm) and 7.5 $\mu g/m^3$ (0.004 ppm), respectively. The measurements cited for North Carolina and Pike's Peak indicate that background concentrations of NO and NO_2 combined can range from 0.001 to 0.005 ppm in remote areas of the United States (Robinson and Robbins, 1972).

More recent measurements using modern methods have yielded results pointing to lower values. Noxon (1975) reports lower tropospheric NO_2 concentrations at a remote site in Colorado mountains of up to 0.20 $\mu g/m^3$ (0.0001 ppm) measured by ground-based absorption spectroscopy. In a more extensive study, Noxon (1978) reports detailed background measurements at the Colorado site and at a number of other widely dispersed locations using methodology with a sensitivity of 0.03 $\mu g/m^3$ (0.015 ppb) at sea level. The author concludes that in the truly unpolluted troposphere the column abundance of NO_2 is less than 5×10^{14} molecules/cm^2. Assuming an effective length for the NO_2 column of about 2 km (Chameides, 1975; Crutzen et al., 1978), this implies a ground level NO_2 background concentration of less than 0.3 $\mu g/m^3$ (0.15 ppb). If the length of the column is 0.5 km (Ritter et al., 1979), background NO_2 concentrations of 0.94 $\mu g/m^3$ (0.5 ppb) would be implied. In addition, Noxon reports that the ground level NO_2 concentration produced at distances greater than 50 km from urban centers seldom exceeds that for truly unpolluted air by more than a factor of 2. Measurements by

Ritter et al. (1979) using chemiluminescence analyzers at a site in Michigan showed NO_x long-term average concentrations in presumably clean air coming from Canada to be in the range of 0.56 to 0.94 μg/m^3 (0.3 to 0.5 ppb). Measurements made at the Colorado site led Ritter et al. to conclude that tropospheric NO_x is confined to the lower 0.5 km of the troposphere. Table 8-1, taken directly from Ritter et al., summarizes background NO_x measurements.

TABLE 8-1. BACKGROUND NO_x MEASUREMENTS (RITTER ET AL., 1979)

Author	Location	Date	NO_x Concentration Observed, ppb
Junge (1956)	Florida	1956	1.0-2.0
Lodge and Pate (1966)	Panama	1966	0.5
Breeding et al. (1973)	Central U.S.	1973	1.0-3.0
Moore (1972)	Boulder, CO	1974	0.1-0.3
Drummond (1976)	Wyoming	1976	0.1-0.4
Cox (1977)	Ireland	1977	0.2-2.0
Galbally (1977)	S. Australia	1977	0.1-0.5
Ritter et al. (1979)	Rural MI	1977	0.3-0.5
	Fritz Peak, CO	1977	0.2 and up

8.1.2 Ambient Concentrations of NO_x

8.1.2.1 Monitoring for NO_x--Data from stationary monitoring sites may be used to estimate the exposure experienced by nearby receptors. The air arriving at a fixed observation point at any time has a unique history. The aspects of this history which determine the ambient concentrations and relative amounts of nitrogen oxides are the sources encountered along the trajectory and a variety of meteorological variables. Atmospheric reactions, such as those that oxidize NO to NO_2, are functions of the concentrations of pollutants emitted to the atmosphere, temperature, sunlight, and time. Other meteorological factors, such as wind speed, vertical temperature structure, and the region's topography, affect the dispersion and dilution of both the directly emitted pollutants and the products of atmospheric reactions.

Given the complex nature of the processes which give rise to potential human exposures, one practical means of estimating these exposures is by monitoring atmospheric concentrations. Air monitoring data relevant to assessing ambient levels of NO_x or NO_x-derived pollutants are collected to meet a variety of specific objectives including:

- Determination of current air quality and trend analysis
- Determination of the state of attainment of National Ambient Air Quality Standards
- Preparation of environmental impact statements
- Development of effective control strategies and evaluation of their effectiveness
- Development and validation of mathematical models which relate the strength of source emissions to predicted concentrations for a variety of meteorological and topographic conditions
- Research, such as studies of the effects of ambient air pollution on human health and welfare.

In general, each specific objective requires special consideration as to site location, frequency and techniques of sampling, and the total amount of data collected. For example, several years of NO_2 data from a number of strategically located sites distributed nationwide might be required for national pollutant trends analysis. A greater number of sites, also collecting data on a regular basis throughout the year, might be necessary for determining compliance with the National Ambient Air Quality Standards. In contrast, only a few carefully chosen days of detailed measurements of various pollutant concentrations and emissions, and meteorological parameters as well, might suffice for validation of mathematical air quality models. The ambient air quality data reported in this chapter are mainly related to the first of the above objectives.

Once an air monitoring station's location is chosen, additional practical considerations arise relating to the actual placement of probes for sampling ambient air. Building surfaces and other obstacles may possibly scavenge NO_2 from ambient air. For this reason, probes must be located a certain minimum distance away from such obstacles. It is important, also, that the oxides of nitrogen in the parcel of air sampled have had sufficient time to undergo atmospheric chemical reactions (such as conversion of NO emissions to NO_2) characteristic of the polluted atmosphere. For this reason, and to avoid sampling air dominated by any one source, probes must also be located some minimum distance from primary sources. Siting considerations have been reviewed in more detail by Ludwig and Shelar (1978); and EPA has proposed guidelines for air quality surveillance and data reporting which include more detailed discussion of considerations noted briefly in this section (Federal Register, August 7, 1978).

8.1.2.2 Sources of Data--The emphasis in monitoring NO_x has been primarily on NO_2, since it is the only nitrogen oxide for which a National Ambient Air Quality Standard has been set. The most complete collection of monitoring data for the United States is EPA's National Aerometric Data Bank (NADB), which receives data from a variety of Federal, state, and local air monitoring programs. The analytical methods with sufficient temporal resolution to assess short-term exposures are continuous versions of the Griess-Saltzman method (e.g., the Lyshkow-modified Griess-Saltzman) and chemiluminescence. These data are reported as 1-hr average concentrations and are archived in SAROAD (Storage and Retrieval of Aerometric Data) a computer-assisted storage and retrieval system of EPA's NADB. Other data, using 24-hr instrumental averaging, are also available in SAROAD. Most of these data were obtained using the sodium arsenite method. Most frequently, these data are not collected every day but on a regular schedule yielding 24-hr measurements, typically once every 6 days.

Another important source of data in recent years, albeit only from the greater St. Louis area, is the intensive monitoring activity carried out as part of the Regional Air Pollution Study (RAPS). Nitrogen dioxide data from this study are routinely available on an hourly basis (minute-by-minute data are also available) as obtained with chemiluminescence monitors at 25 Regional Air Monitoring Sites (RAMS). These data are archived in a special RAPS data base maintained at EPA's Research Triangle Park Environmental Research Center.

8.1.3 Historical Measurements of NO_x Concentrations

In past years, the EPA Continuous Air Monitoring Program (CAMP) provided the data set covering the longest period of time on both NO and NO_2 concentrations available in this country. Caution is necessary in using these data because collection and reporting procedures were not subject to detailed quality assurance checks and more than one operation and maintenance procedure may have been used over the years at a given site. However, the data base presented here does not include data taken by the Jacobs-Hochheiser method, because this method has been withdrawn by EPA (Chapter 7). The data collected provide a useful historical perspective on trends of NO_x concentrations. Table 8-2 presents 12 years of measurements of nitrogen oxide (NO) at 6 CAMP stations for the time period 1962-1973. The annual average NO concentrations are plotted in Figure 8-1. Trends in concentrations during 1962-1973 were generally upward for all sites monitored. When the annual means are grouped by 5-year periods, 1962-1966 and 1967-1971 (Table 8-3), both the second highest value and the annual means averaged over all CAMP cities showed an increase of about 15 percent from the earlier to later time period. Similar, though more geographically variable, results obtained for nitrogen dioxide (NO_2) concentrations at most CAMP sites (Table 8-4, 8-5; Figure 8-2). St. Louis data showed a marked decrease, however, in both annual average concentrations and average of second highest value between the two 5-year periods (Table 8-5). Upward trends in annual average NO_2 concentrations were observed in 3 of the 5 CAMP cities over the 9-year period, 1963 to 1971. Figure 8-3 is a graphical presentation of changes in NO_2 air quality in the Los Angeles basin between the years 1965 and 1974. The average increase in annual means for 11 stations in the basin was about 20 percent, but individual area results varied widely, as seen in Figure 8-3.

TABLE 8-2. YEARLY AVERAGE AND MAXIMUM CONCENTRATIONS OF NITRIC OXIDE AT CAMP STATIONS, MEASURED BY THE CONTINUOUS SALTZMAN COLORIMETRIC METHOD (U.S. EPA, 1975a)

	Concentration, μg/m³ (ppb), 25°C											
	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
Year	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1962	-- --	-- --	37 (30)	788 (630)	123 (98)	739 (704)	-- --	-- --	37 (30)	702 (562)	25 (20)	431 (345)
1963	-- --	-- --	49 (39)	1,060 (848)	123 (98)	615 (492)	-- --	-- --	37 (30)	615 (492)	62 (50)	1,845 (1,476)
1964	-- --	-- --	49 (39)	1,070 (856)	123 (98)	1,105 (884)	49 (39)	923 (738)	49 (39)	787 (630)	62 (50)	1,400 (1,120)
1965	37 (30)	652 (522)	37 (30)	751 (600)	123 (98)	750 (600)	37 (30)	443 (354)	37 (30)	750 (600)	62 (50)	1,083 (866)
1966	49 (39)	627 (502)	49 (39)	1,240 (1,000)	123 (98)	775 (620)	37 (30)	688 (550)	49 (39)	1,230 (984)	74 (60)	2,290 (1,832)
1967	49 (39)	590 (472)	62 (50)	1,390 (1,112)	98 (78)	763 (610)	49 (39)	393 (314)	37 (30)	1,685 (1,348)	74 (60)	1,820 (1,456)
1968	49 (39)	738 (590)	49 (39)	837 (670)	86 (77)	739 (591)	37 (30)	492 (394)	74 (60)	1,242 (994)	62 (50)	1,735 (1,388)
1969	49 (39)	677 (542)	49 (39)	959 (767)	135 (108)	1,920 (1,536)	37 (30)	873 (698)	49 (39)	861 (689)	49 (39)	1,083 (866)

(continued)

TABLE 8-2. (continued)

Year	Concentration, $\mu g/m^3$ (ppb), $25^{0}C$											
	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1970	62 (50)	750 (600)	62 (50)	1,430 (1,144)	172 (137)	2,240 (1,792)	62 (50)	689 (551)	49 (39)	960 (768)	74 (60)	1,672 (1,338)
1971	62 (50)	677 (542)	49 (39)	775 (620)	135 (108)	824 (659)	62 (50)	615 (492)	62 (50)	750 (600)	49 (39)	935 (748)
1972	74 (60)	788 (630)	86 (69)	825 (660)	160 (128)	787 (630)	62 (50)	714 (571)	49 (39)	763 (610)	62 (50)	800 (640)
1973	74 (60)	652 (522)	123 (98)	640 (512)	221 (177)	775 (620)	74 (60)	750 (600)	49 (39)	689 (551)	-- --	-- --

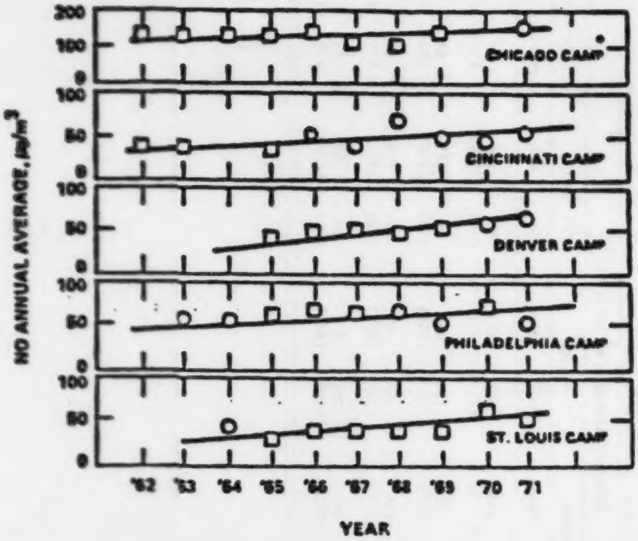

Figure 8-1. Trend lines for nitric oxide annual averages in five CAMP cities. —◻— :data satisfying NADB minimum sampling criteria; —○— : invalid average (based on incomplete data). *Note change in ordinate scale for these data (U.S. EPA, 1973).

TABLE 8-3. FIVE-YEAR AVERAGES OF NITRIC OXIDE CONCENTRATIONS AT CAMP STATIONS, MEASURED BY CONTINUOUS SALTZMAN COLORIMETRIC METHOD (U.S. EPA, (1973)

Station	Average Concentration, $\mu g/m^3$ (ppb), 25°C			Average of Annual 2nd Highest Value, $\mu g/m^3$ (ppb), 25°C		
	1962-1966	1967-1971	Change, %	1962-1966	1967-1971	Change, %
Chicago	122.6 (98.1)	125.4 (100.3)	+ 2	731 (584.8)	969 (775.2)	+32
Cincinnati	43.8 (35.0)	53.6 (42.9)	+22	782 (625.6)	1,067 (853.6)	+36
Denver	44.9 (35.9)	54.4 (43.52)	+21	633 (506.4)	620 (496.0)	- 2
Philadelphia	55.2 (44.2)	65.4 (52.3)	+18	1,331 (1,064.8)	1,395 (1,116.0)	+ 5
St. Louis	39.8 (31.8)	47.6 (38.1)	+19	541 (287.8)	578 (462.4)	+ 7
CAMP average	61.2 (49.0)	69.3 (55.4)	+13	804 (643.2)	926 (740.8)	+15

TABLE 8-4. YEARLY AVERAGE AND MAXIMUM CONCENTRATIONS OF NITROGEN DIOXIDE AT CAMP STATIONS, MEASURED BY THE CONINUOUS SALTZMAN COLORIMETRIC METHOD (U.S. EPA, 1975a)

Year	Concentration, $\mu g/m^3$, at 25°C											
	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1962	--	--	56	545	75	394	--	--	56	470	38	226
1963	--	--	56	394	75	376	--	--	56	377	75	504
1964	--	--	75	413	94	865	56	394	56	620	75	471
1965	75	507	56	770	75	319	56	226	56	301	75	358
1966	56	602	75	319	113	564	56	357	75	432	75	413
1967	75	583	75	376	94	470	38	282	56	432	75	413
1968	75	488	94	432	94	319	38	319	56	1,036	75	358
1969	56	620	75	432	94	358	56	714	56	394	75	245
1970	75	639	94	545	113	395	56	244	75	433	94	377
1971	75	676	75	413	113	1,090	56	244	56	301	75	770
1972	75	583	113	1,202	94	413	94	376	75	319	75	545
1973	94	846	--	--	132	676	56	508	75	207	--	--

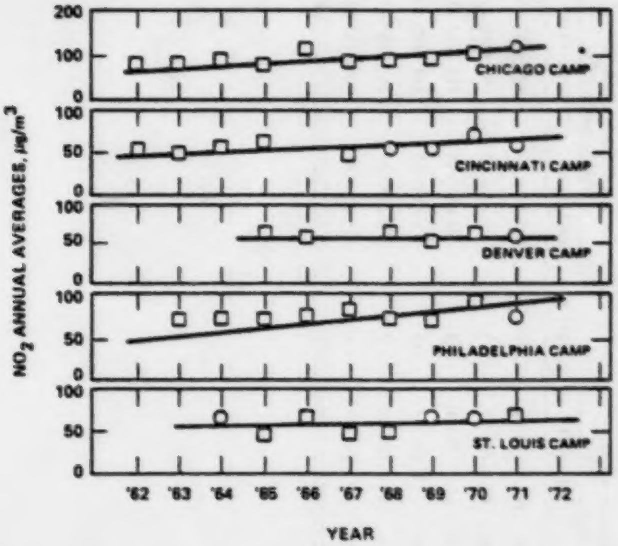

Figure 8-2. Trend lines for nitrogen dioxide annual averages in five CAMP cities. –□– : data satisfying NADB minimum sampling criteria; –○– : invalid average (based on incomplete data). *Note change in ordinate scale for these data (U.S. EPA, 1973).

TABLE 8-5. FIVE-YEAR AVERAGES OF NITROGEN DIOXIDE CONCENTRATIONS AT CAMP STATIONS, MEASURED BY THE CONTINUOUS SALTZMAN COLORIMETRIC METHOD (U.S. EPA, 1973)

Station	Average Concentration, µg/m³ (ppb), 25°C		Change, %	Average of Annual 2nd Highest Value, µg/m³ (ppb), 25°C		Change, %
	1962-1966	1967-1971		1962-1966	1967-1971	
Chicago	86.1 (45.8)	101.2 (53.8)	+18	444 (236.2)	499 (265.4)	+12
Cincinnati	62.0 (33.0)	60.0 (31.9)	- 3	391 (208.0)	367 (195.2)	- 6
Denver	66.0 (35.1)	67.9 (36.1)	+ 3	498 (264.9)	493 (262.2)	- 1
Philadelphia	67.7 (36.0)	77.6 (41.3)	+15	361 (192.0)	414 (220.2)	+15
St. Louis	58.5 (31.1)	54.2 (28.8)	- 7	320 (170.2)	267 (142.0)	-16
CAMP average	68.1 (36.2)	72.2 (38.4)	+ 6	403 (214.4)	408 (217.0)	+ 1

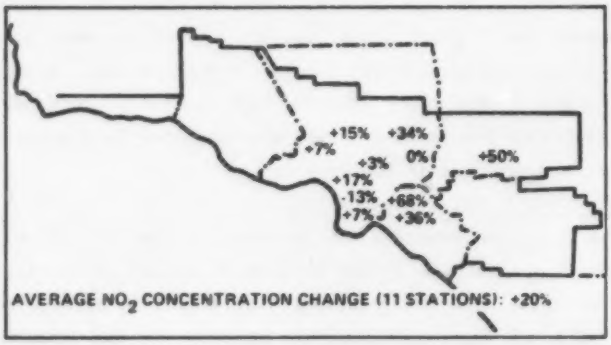
+15%
+34%
+7%
+3%
0%
+50%
+17%
-13%
+68%
+7%
+36%
AVERAGE NO_2 CONCENTRATION CHANGE (11 STATIONS): +20%

Figure 8-3. Trends in NO_2 air quality, Los Angeles Basin, 1965-1974 (Trijonis et al., 1976).

8.1.4 Recent Trends in NO_2 Concentrations

Examination of data on trends in NO_2 concentrations during more recent years presents a variable picture at selected sites across the nation (Figures 8-4 through 8-9). Since measured concentrations, particularly those dealing with short-term peak excursions, may be expected to depend upon specific site considerations as well as varying meteorological parameters, the only data presented are those from the same site over the years plotted. Consistent downward trends are observed at Camden, New Jersey, for all statistics presented (Figure 8-4). A generally downward trend for peak one-hour levels is also discernible at a site in downtown Los Angeles (Figure 8-5), accompanied by a relatively level pattern for other statistics plotted; and in Azusa, California (Figure 8-6), similar generally level trends are apparent for all statistics plotted for 1971-1980. Nitrogen dioxide air quality seems to have steadily improved in Newark, New Jersey (Figure 8-7) from 1971 to 1977, the last year of available NO_2 data at the Newark site plotted. No clearcut trend is discernible in Portland, Oregon (Figure 8-8), although an unusually high one-hour peak level was recorded in 1979 during a day on which several other air pollutants (such as CO) were also markedly elevated over usual levels. Figure 8-9 shows trends in a 4-year running average of annual averages (during 1970-1975) of daily maximum 1-hr NO_2 concentrations in the Los Angeles basin. A marked decrease in both the highest annual average and in the mean of 5 sites is apparent, although the lowest annual average increased over the same period. By comparison (Table 8-6), NO_2 concentrations spanning the years 1969 to 1974 generally increased for nearby sites in rapid growth areas of Orange County California, whereas other California sites listed in Table 8-6 generally experienced declines in NO_2 levels.

8.1.5 Seasonal Variations in NO_2 Concentrations

In this section, a few examples of seasonal variations in NO_2 concentrations are presented which demonstrate that no single nationwide pattern exists for the monthly averages of daily maximum 1-hr data. The month-to-month variations in average NO_2 concentrations may be the combined result of high photochemical activity in the summer months, time-varying emissions of NO_x (with high emissions of NO_x during the winter in some areas), time-varying emissions of hydrocarbons, and area-specific meteorological conditions throughout the year.

The data presented are, for the most part, averaged over several years of monitoring, a procedure which may be expected to yield representative patterns for the regions reported. Examination of Figures 8-10 and 8-11 reveals that Chicago, Illinois, experiences a marked peak in NO_2 concentrations during the summer months while concentrations in Denver, Colorado, appear to peak in the winter. In contrast to the above patterns, certain sites in southern California (Los Angeles, Azusa, and Pomona) do not exhibit a marked seasonality. (The pattern for Lennox, as publish in Trijonis, 1978, was discovered to be a duplicate of the Denver pattern and is corrected here).

8.1.6 Recently Observed Atmospheric Concentrations of NO_2

In this section, representative examples of observed concentrations of NO_2 in recent years are presented. In summary, the data cited illustrate the following points:

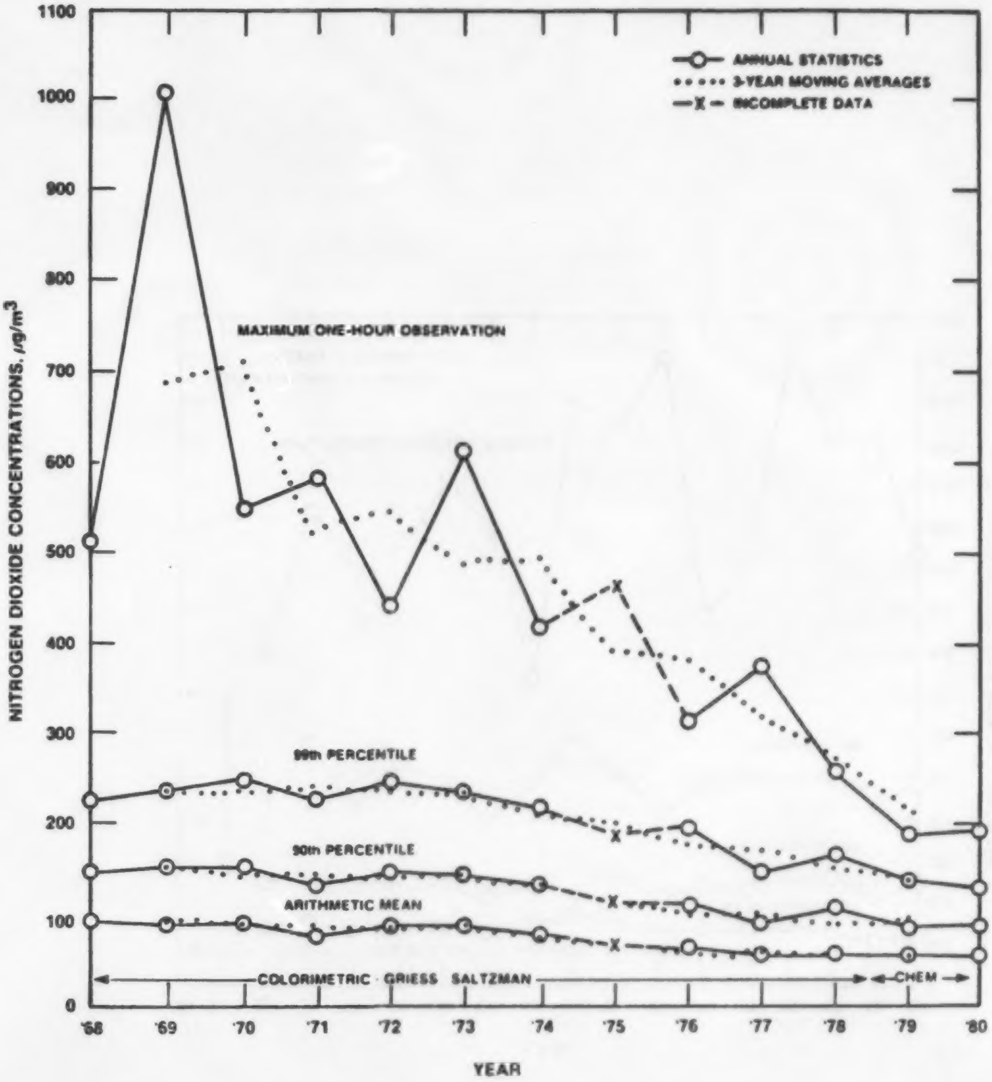

Figure 8-4. Annual air quality and 3-year moving averages at Camden, New Jersey. [Data adapted from Trijonis [1978], augmented with data from SAROAD.]

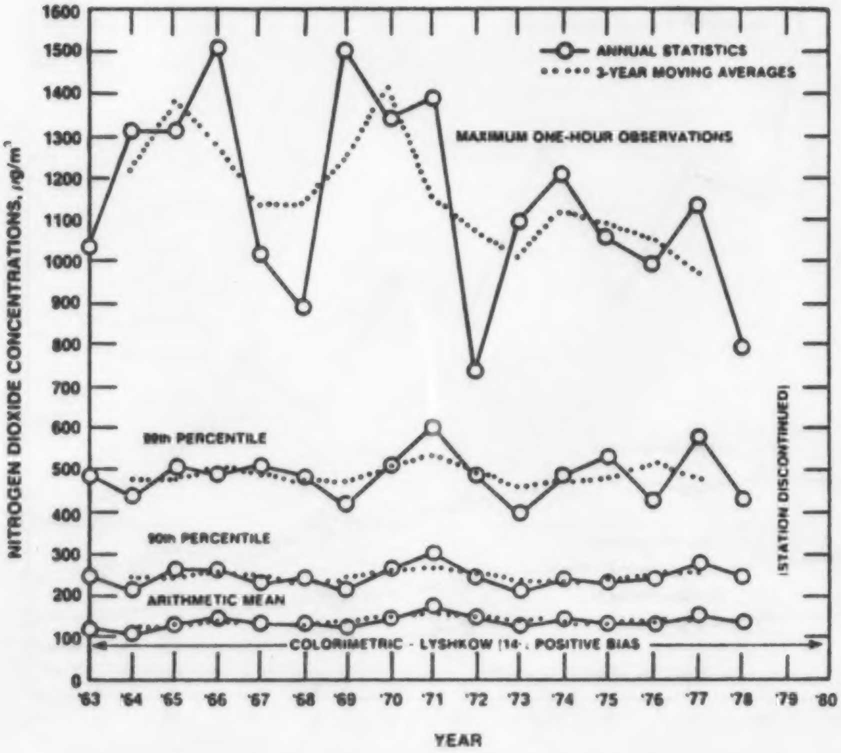

Figure 8-5. Annual air qualtiy statistics and 3-year moving averages at downtown Los Angeles. California. [Data adapted from Trijonis [1978], augmented with data from SAROAD.]

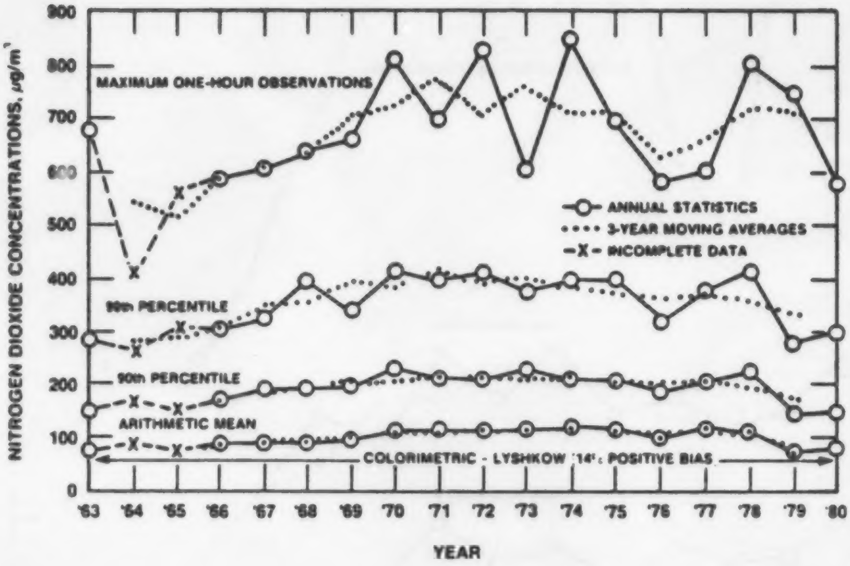

Figure 8-6. Annual air quality statistics and 3-year moving averages at Azusa, California. [Data adapted from Trijonis [1978], augmented with data from SAROAD.]

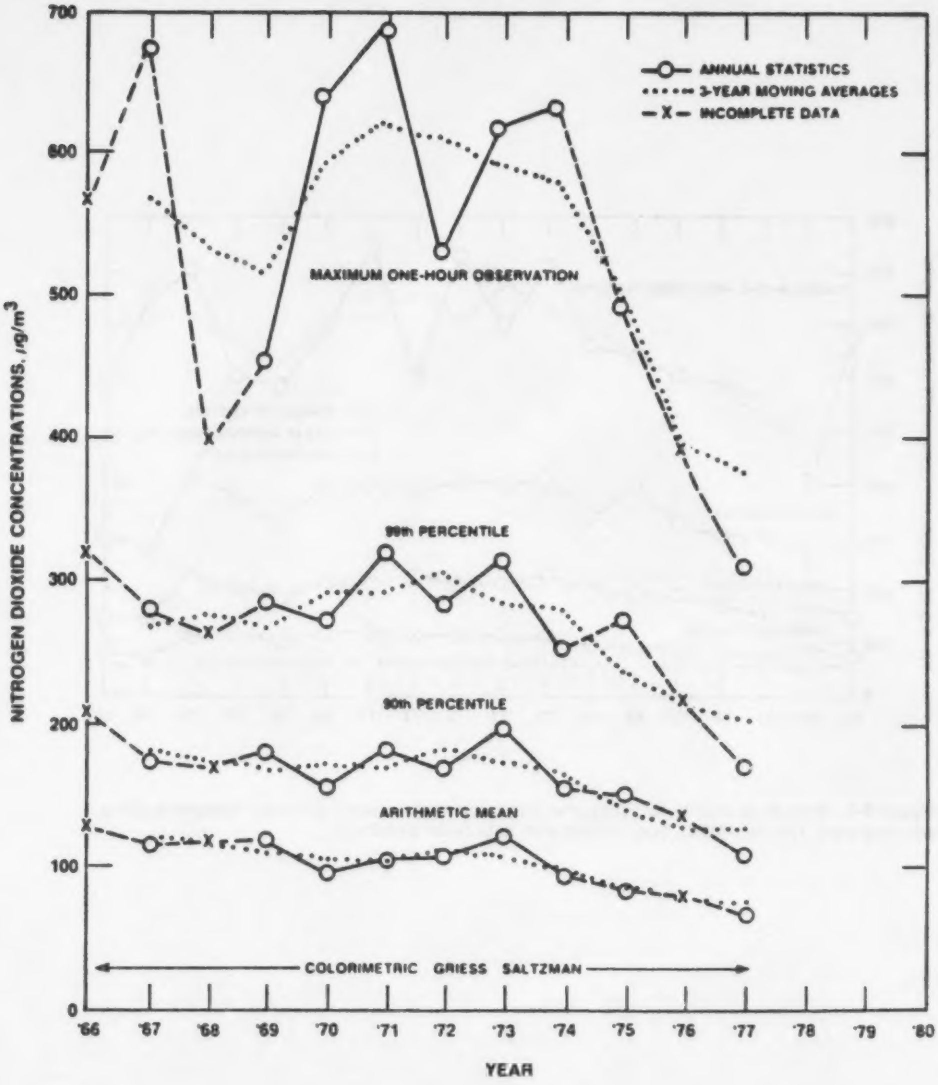

Figure 8-7. Annual air quality statistics and 3-year moving averages at Newark, New Jersey. [Data adapted from Trijonis [1978], augmented with data from SAROAD.]

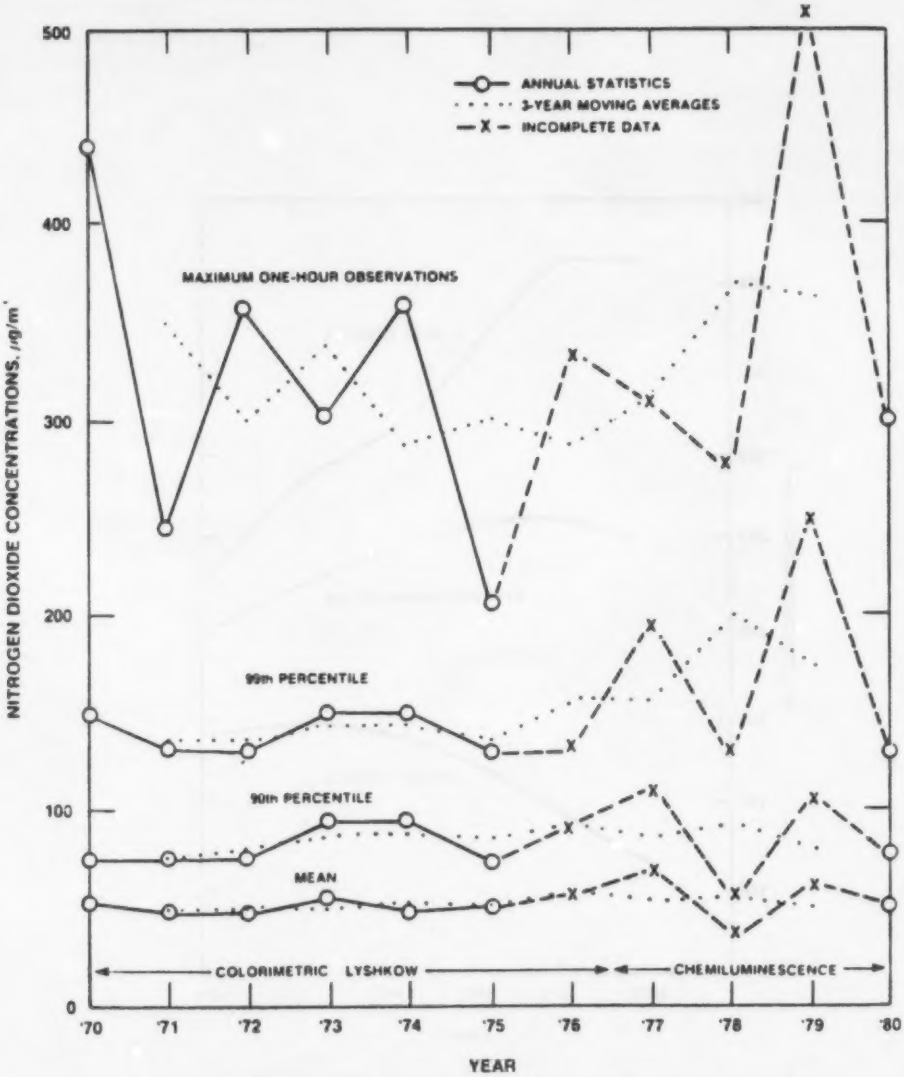

Figure 8-8. Annual air quality statistics and 3-year moving averages at Portland, Oregon. [Data adapted from Trijonis [1978], augmented with data from SAROAD.]

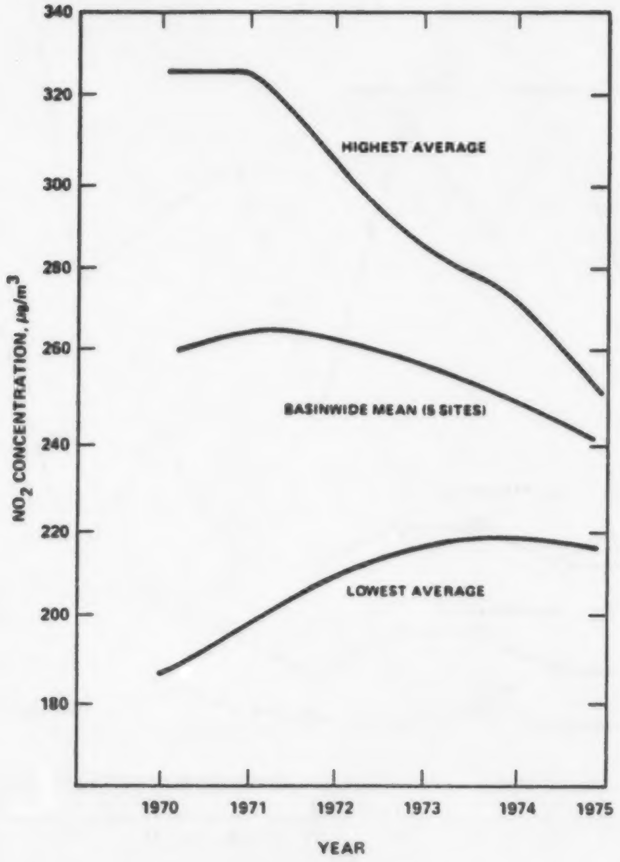

Figure 8-9. Annual average of daily maximum 1-hour NO_2 (4-year running mean) in the Los Angeles Basin (U.S. EPA, 1976a).

TABLE 8-6. FIVE-YEAR CHANGES IN AMBIENT NO_2 CONCENTRATIONS[a]

		NET PERCENTAGE CHANGE IN NO_2 CONCENTRATIONS FROM 1969 TO 1974		
STATIONS		Annual Mean	90th Percentile	Yearly Maximum
LOS ANGELES BASIN SITES				
Orange County:	Anaheim	+ 9%	+ 5%	+13%
(rapid growth)	La Habra	+99%	+60%	+72%
Average for Orange County		+54%	+33%	+43%
Los Angeles County:	Azusa	+17%	+ 7%	+ 6%
(slow growth)	Lennox	- 7%	-11%	+ 1%
	Los Angeles	- 5%	- 2%	-28%
	L.A. (Westwood)	+ 8%	+11%	+32%
	L.A. (Reseda)	- 4%	-10%	-13%
Average for Los Angeles County		+ 3%	- 1%	- 1%
OTHER CALIFORNIA SITES				
	Oakland	- 7%	- 9%	-14%
	Pittsburg	- 8%	- 4%	-12%
	Redwood City	-24%	-25%	- 9%
	Salinas	- 1%	- 1%	+27%
	San Rafael	+ 5%	0%	0%
	Santa Cruz	+15%	-24%	-27%
	Stockton	- 3%	-44%	-21%
Average for Other California Sites		- 3%	-15%	- 8%
NEW JERSEY SITES				
	Bayonne	-27%	-18%	-36%
	Camden	- 9%	- 7%	-52%
	Newark	+ 2%	0%	0%
Average for New Jersey Sites		-14%	- 8%	-24%
OTHER SITES				
	Chicago, IL	+32%	+51%	+94%
	Portland, OR	-.7%	+44%	- 3%

[a] Adapted from Trijonis (1978).

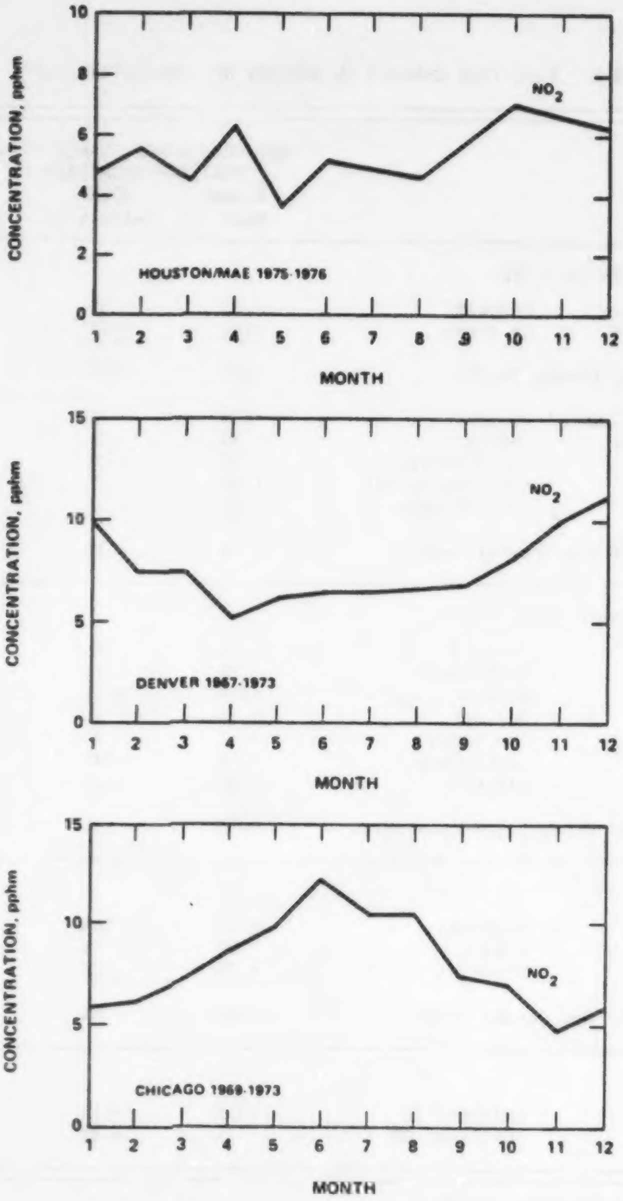

Figure 8-10. Seasonal NO$_2$ concentration patterns of three U.S. urban sites (monthly averages of daily maximum 1-hr concentrations). Adapted from Trijonis (1978).

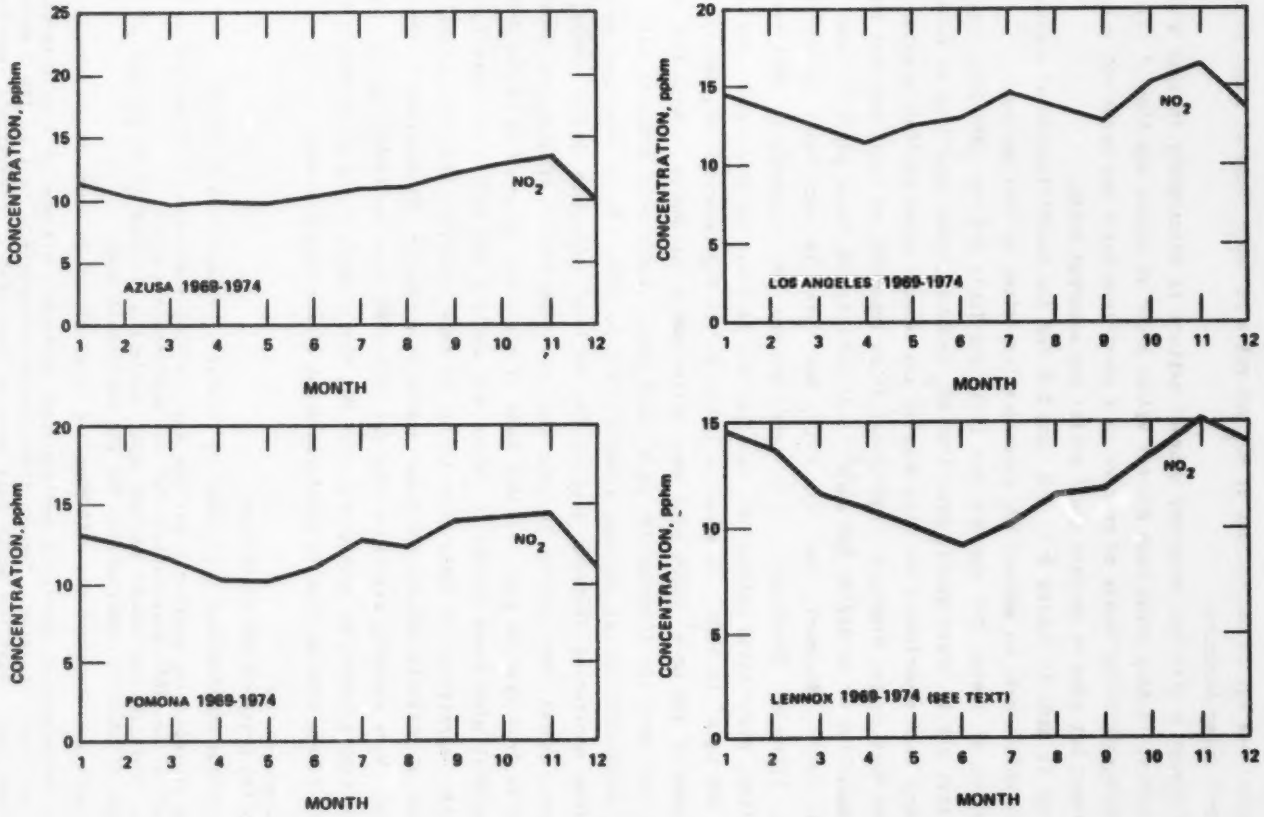

Figure 8-11. Seasonal NO_2 concentration patterns of four U.S. urban sites (monthly averages of daily maximum 1-hr concentrations). Adapted from Trijonis (1978).

- Annual average concentrations of NO_2 are not a reliable index of short-term (3-hr or less) human exposure.
- Although a distinct recurrent diurnal pattern is discernible in some areas of the country, in many areas peak diurnal values occur at almost any time of day.
- Nitrogen dioxide levels of concern on a short-term basis may occur not only in urban areas, but also in certain small cities and suburban areas.

Reference is made to Tables 8-7, 8-8, and 8-9 for the identification of analytic procedures used to obtain data on ambient NO_2 concentrations cited in this section.

Examination of Tables 8-7 and 8-8 for 1975, and Table 8-9 for 1976-1980, reveals that during at least one of these years, peak 1-hr NO_2 concentrations equalling or exceeding 750 $\mu g/m^3$ (0.4 ppm) were experienced in: Los Angeles and several other California sites; Ashland, Kentucky; and Port Huron, Michigan. Additional sites reporting at least one peak hourly concentration equalling or exceeding 500 $\mu g/m^3$ (0.27 ppm) during those years include: Phoenix, Arizona; St. Louis, Missouri; New York City, New York; 14 additional California sites; Springfield, Illinois; Cincinnati, Ohio; and Saginaw and Southfield, Michigan. Other scattered sites, distributed nationwide, reported maxima close to this value, including some approaching 500 $\mu g/m^3$ in 1980. As shown in Tables 8-7 to 8-9 recurrent NO_2 hourly concentrations in excess of 250 $\mu g/m^3$ (0.14 ppm) were quite common nationwide in both 1975 and subsequent years, but very few exceeded 750 $\mu g/m^3$ (0.4 ppm). Table 8-10 presents data for 24-hr average NO_2 concentrations at various sites in 1976 to 1980. It is important to note that only data from monitoring stations meeting EPA National Air Data Branch (NADB) sampling criteria* were chosen for listing in this section. The number of stations meeting these criteria varies from year to year, so that many of the areas reported in Tables 8-9 and 8-10 are not identical with those listed in Tables 8-7 and 8-8 for 1975. The data for the wide range of areas represented in these four tables do show, however, that occassional peak NO_2 concentrations of possible concern for human health (see Chapter 15) occurred in the nation in the mid-1970s. More recently available data for 1976-1980 from the SAROAD system suggest that basically the same patterns of occassional peak NO_2 levels approaching or exceeding 0.4 to 0.5 ppm still occur from time to time in scattered areas of the United States.

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* NADB sampling criteria are as follows:

1) For continuous observations with sampling intervals of less than 24 hours:
   a) Data representing quarterly periods must reflect a minimum of 75 percent of the total number of possible observations for the applicable quarter.
   b) Data representing annual periods must reflect a minimum of 75 percent of the total number of possible observations for the applicable year.
2) For noncontinuous observations with sampling intervals of 24 hours or greater:
   a) Data representing quarterly periods must reflect a minimum of five observations for the applicable quarter. Should there be no measurements in 1 of the 3 months of the quarter, each remaining month must have no less than 2 observations reported for the applicable period.
   b) Data representing annual periods must reflect four quarters of observation that have satisfied the quarterly criteria.

TABLE 8-7. RATIO OF MAXIMUM OBSERVED HOURLY NITROGEN DIOXIDE CONCENTRATIONS TO ANNUAL MEANS DURING 1975 FOR SELECTED LOCATIONS*

		$\mu g/m^3$ Maximum hourly concentration - yearly arithmetic mean	
State	Location	Method A[b]	Method B[c]
California	Anaheim	940/101= 9.3	-
	Azusa	696/112= 6.2	-
	Costa Mesa	658/58 =11.3	-
	Los Angeles[a]	1053/126= 8.4	-
		1128/129= 8.7	-
	Lynwood	602/97 = 6.2	-
	San Bernadino	470/76 = 6.2	-
	Napa	188/49 = 3.8	-
	San Francisco	432/62 = 7.0	-
	Barstow	432/39 =11.1	-
	Fontana	-	489/78 = 6.3
	Chula Vista	-	451/64 = 7.1
	Visalia	-	226/48 = 4.7
Colorado	Denver[a]	-	555/96 = 5.8
Georgia	Atlanta	-	489/76 = 6.4
Illinois	Chicago[a]	-	395/104= 3.8
		-	395/109= 3.6
		-	244/41 = 6.0
	East St. Louis	-	293/65 = 4.5
Kentucky	Paducah	714/66 =10.8	-
	Louisville[a]	-	348/84 4.1
	Ashland	895/85 =10.5	-

(continued)

TABLE 8-7. (continued)

		$\mu g/m^3$ Maximum hourly concentration - yearly arithmetic mean	
State	Location	Method A[b]	Method B[c]
Maine	Bangor	270/49 = 5.5	-
Maryland	Essex	-	282/53 = 5.3
Michigan	Grand Rapids	279/67 = 4.2	-
	Detroit	-	338/58 = 5.8
Oregon	Portland	207/50 = 4.1	-
Texas	Dallas	-	432/32 =13.5

[a] More than one station reporting.

[b] Method A: Instrumental Colorimetric-Lyshkow (MOD) method, a variation of the continuous Greiss-Saltzman Method.

[c] Method B: Instrumental Chemiluminescence Method.

*For comparison purposes, note that: 1.0 ppm NO_2 ≅ 1880 $\mu g/m^3$; 0.5 ppm ≅ 940 $\mu g/m^3$; 0.1 ppm ≅ 188 $\mu g/m^3$; 0.05 ppm ≅ 94 $\mu g/m^3$; and 0.01 ppm ≅ 18.8 $\mu g/m^3$.

TABLE 8-8. FREQUENCY DISTRIBUTION OF 1975 HOURLY NO_2 CONCENTRATIONS AT VARIOUS SITES IN U.S. URBAN AREAS (U.S. EPA, 1977a)*

Location	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations				Maximum observed concentration ($\mu g/m^3$)
	1%	5%	10%	50%	
Arizona					
Phoenix[a]	271	188	152	69	660
California					
Los Angeles[a]	526	301	226	94	1053
Redlands[a]	282	169	132	56	545
Redlands[b]	226	150	132	56	357
Riverside[a]	301	226	188	94	564
Riverside[b]	395	282	226	113	658
San Diego[a]	226	150	113	38	508
San Diego[b]	395	282	226	113	865
Colorado					
Denver[a]	282	188	150	94	432
Denver[b]	265	177	149	90	483
Kentucky					
Ashland[a]	297	209	173	68	895
Michigan					
Detroit[b]	150	113	94	56	338
Missouri					
St. Louis[a]	338	244	207	75	658
New Jersey					
Newark[c]	273	169	150	81	494
Newark[b]	226	169	132	75	376
New York					
New York City[b]	226	169	132	56	526
Ohio					
Cincinnati[b]	282	150	103	47	395
Pennsylvania					
Philadelphia[b]	301	226	207	113	451
Texas					
Dallas[b]	132	94	75	19	432

[a] Obtained by Instrumental Colorimetric-Lyshkow (Mod) method, a variation of the Griess-Saltzman method.

[b] Obtained by Chemiluminescence Method.

[c] Obtained by Instrumental Colorimetric-Griess-Saltzman method.

*For comparison purposes, note that: 1.0 ppm $NO_2 \cong 1880\ \mu g/m^3$; 0.5 ppm $\cong 940\ \mu g/m^3$; 0.1 ppm $\cong 188\ \mu g/m^3$; 0.05 ppm $\cong 94\ \mu g/m^3$; and 0.01 ppm $\cong 18.8\ \mu g/m^3$.

TABLE 8-9. FREQUENCY DISTRIBUTION OF 1976, 1978, AND 1980 HOURLY NITROGEN DIOXIDE CONCENTRATIONS AT VARIOUS U.S. SITES (U.S. EPA, 1976c, 1979, 1981)[a]

Location	SAROAD Site ID	Method	Year	Concentrations equalled or exceeded by stated percent of observations ($\mu g/m^3$) 1%	10%	50%	Maximum observed concentration ($\mu g/m^3$)	2nd Highest observed concentration ($\mu g/m^3$)	Yearly arithmetic mean ($\mu g/m^3$)
Arizona									
Phoenix	030600002G01	Instrumental	1976	226	150	56	451	432	60
		Chemiluminescence	1978	150	75	38	226	207	44
			1980	94	38	5	132	132	17[b]
Tucson	030860002G01	Instrumental	1976	132	94	56	451	357	51
		Chemiluminescence	1978	150	100	56	432	301	61[b]
			1980	301	150	56	414	414	69
California									
Anaheim	050230001I01	Griess-Saltzman[a]	1976	395	188	94	865	752	103
		(Lyshkow)	1978	338	150	75	564	545	90[b]
			1980	226	132	56	470	395	71[b]
Chino	051300001I01	Instrumental	1976	414	132	19	602	583	54
		Chemiluminescence	1978	226	150	94	301	282	94[b]
			1980	-	-	-	-	-	-
Costa Mesa	051740002I01	Griess-Saltzman[a]	1976	301	132	19	639	639	53
		(Lyshkow)	1978	263	113	38	564	545	53[b]
			1980	263	113	38	583	526	50[b]
El Cajon	055300002I01	Instrumental	1976	320	188	94	545	489	108
		Chemiluminescence	1978	301	169	94	508	432	104[b]
			1980	244	169	94	338	282	95[b]
Fontana	052680001I01	Instrumental	1976	244	132	56	564	508	63
		Chemiluminescence	1978	-	-	-	-	-	-
			1980	207	132	56	470	432	73[b]
Fremont	052780001I01	Griess-Saltzman[a]	1976	301	150	56	526	526	80
		(Lyshkow)	1978	207	113	56	320	282	66
			1980	-	-	-	-	-	-
La Habra	053620001I01	Griess-Saltzman[a]	1976	320	169	75	526	508	91
		(Lyshkow)	1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-

TABLE 8-9. (continued)

Location	SAROAD Site ID	Method	Year	Concentrations equalled or exceeded by stated percent of observations ($\mu g/m^3$) 1%	10%	50%	Maximum observed concentration ($\mu g/m^3$)	2nd highest observed concentration ($\mu g/m^3$)	Yearly arithmetic mean ($\mu g/m^3$)
California (cont.)									
Oakland	055300004F01	Instrumental Chemiluminescence	1976	301	150	56	545	489	77
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
Oceanside	055320003I01	Instrumental Chemiluminescence	1976	263	113	38	620	620	57
			1978	244	113	56	602	545	58[b]
			1980	207	75	38	357	338	40[b]
Redlands	056200001I01	Instrumental Chemiluminescence	1976	169	75	38	470	451	47
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
Riverside	056400005F01	Instrumental Chemiluminescence	1976	338	207	94	564	526	113[b]
			1978	301	169	94	564	489	101
			1980	282	150	75	414	395	90[b]
San Diego	056800006I01	Instrumental Chemiluminescence	1976	244	113	56	451	451	63
			1978	226	132	56	432	395	74[b]
			1980	226	113	38	357	357	55[b]
San Diego	056800004I01	Instrumental Chemiluminescence	1976	357	188	94	585	564	105
			1978	376	207	94	940	846	112[b]
			1980	320	207	94	470	451	114[b]
San Jose	056980004A05	Instrumental Chemiluminescence	1976	301	169	66	479	461	86
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
San Jose	056980004I01	Griess-Saltzman[a] (Lyshkow)	1976	320	169	75	526	507	86
			1978	244	132	75	414	414	78
			1980	207	132	75	301	°282	79[b]

TABLE 8-9. (continued)

Location	SAROAD Site ID	Method	Year	Concentrations equalled or exceeded by stated percent of observations (μg/m^3)			Maximum observed concentration (μg/m^3)	2nd highest observed concentration (μg/m^3)	Yearly arithmetic mean (μg/m^3)
				1%	10%	50%			
Georgia									
Atlanta	110200001F01	Instrumental Chemiluminescence	1976	160	103	66	244	216	67
			1978	160	113	66	348	292	72[b]
			1980	160	104	56	301	273	61[b]
Illinois									
Chicago	141122001P10	Instrumental Chemiluminescence	1976	273	179	113	461	442	116
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
Chicago	141122002A05	Instrumental Chemiluminescence	1976	263	169	94	461	442	108
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
Springfield	147280003F01	Instrumental Chemiluminescence	1976	90	55	19	519	293	25
			1978	113	70	36	239	205	40[b]
			1980	-	-	-	-	-	-
Kentucky									
Ashland	180080008F01	Griess-Saltzman[a] (Lyshkow)	1976	209	113	55	572	464	63
			1978	141	75	34	306	275	41[b]
			1980	-	-	-	-	-	-
Michigan									
Port Huron	234340003F01	Griess-Saltzman[a] (Lyshkow)	1976	222	121	65	832	815	73
			1978	188	145	71	450	438	79[b]
			1980	-	-	-	-	-	-
Saginaw	234760002F01	Griess-Saltzman[a] (Lyshkow)	1976	291	154	76	643	622	86
			1978	312	169	86	649	629	94[b]
			1980	120	89	45	189	180	48[b]
Southfield	234880002F01	Griess-Saltzman[a]	1976	363	181	83	645	585	100
			1978	292	163	102	365	361	106[b]
			1980	-	-	-	-	-	-

TABLE 8-9. (continued)

Location	SAROAD Site ID	Method	Year	Concentrations equalled or exceeded by stated percent of observations ($\mu g/m^3$) 1%	10%	50%	Maximum observed concentration ($\mu g/m^3$)	2nd highest observed concentration ($\mu g/m^3$)	Yearly arithmetic mean ($\mu g/m^3$)
New Jersey									
Newark	313480002A05	Instrumental Chemiluminescence	1976	226	122	75	338	320	80
			1978	-	-	-	-	-	-
			1980	167	117	66	196	192	70[b]
Ohio									
Cincinnati	361220019A05	Instrumental Chemiluminescence	1976	147	94	56	677	508	60
			1978	150	98	47	254	235	55
			1980	103	70	38	150	145	41
Pennsylvania									
Philadelphia	397140023H01	Greiss-Saltzman[a] (Lyshkow)	1976	188	113	75	451	451	74
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-
Utah									
Salt Lake City	460920001F01	Instrumental Chemiluminescence	1976	244	132	75	470	451	80
			1978	188	113	56	263	263	65[b]
			1980	207	113	56	357	357	61[b]
Salt Lake City	460920001A05	Instrumental Chemiluminescence	1976	226	132	75	470	432	75
			1978	-	-	-	-	-	-
			1980	-	-	-	-	-	-

[a] Data obtained using dynamic calibration procedures.

[b] Data not satisfying NADB minimum sampling criteria.

*For comparison purposes, note that: 1.0 ppm NO ~ 1880 $\mu g/m^3$; 0.5 ppm ~ ppm940 $\mu g/m^3$ 0.1 ppm ~ 188 $\mu g/m^3$; 0.05 ppm ~ 94 $\mu g/m$; and 0.01 ppm ~ 18.8 $\mu g/m^3$.

TABLE 8-10. FREQUENCY DISTRIBUTION OF 1976, 1978, AND 1980 24-HOUR AVERAGE NO_2 CONCENTRATIONS AT VARIOUS SITES IN U.S. URBAN AREAS (ALL DATA OBTAINED BY SODIUM ARSENITE METHOD) (U.S. EPA, 1976c, 1979, 1981)[a]

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations 10%	50%	Annual arithmetic mean ($\mu g/m^3$)
Alabama							
Birmingham	010380003P01	1976	127	117	107	66	69
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Alaska							
Fairbanks	020160001P01	1976	110	103	85	59	59
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Arizona							
Tucson	030860001F01	1976	127	96	69	45	47
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Arkansas							
Little Rock	041440003F01	1976	105	100	65	32	37
	"	1978	93	72	58	28	31
	"	1980	79	76	53	27	30
California							
Fresno	052800002F01	1976	147	133	118	49	58
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Long Beach	054100001F01	1976	339	285	215	101	119
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
San Bernadino	056680001F01	1976	156	154	124	78	85
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Colorado							
Denver	060580001P01	1976	163	140	102	46	55
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations 10%	50%	Annual arithmetic mean ($\mu g/m^3$)
Connecticut							
Bridgeport	070060123F01	1976	143	139	123	66	70
	"	1978	134	130	108	62	66
	"	1980	207	196	114	69	78
Greenwich	070330008F01	1976	101	80	54	33	36
	070060004F01	1978	93	80	65	38	39
	070330004F01	1980	123	101	82	47	52
Florida							
Jacksonville	101960033H01	1976	138	91	90	55	59
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	101960002P01	1976	100	89	79	59	58
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	101960032H01	1976	93	81	67	50	53
	"	1978	50	47	47	34	33[b]
	"	1980	-	-	-	-	-
Orlando	103280004F01	1976	91	76	50	32	32
	"	1978	71	57	47	34	34[b]
	"	1980	74	72	64	40	41
Georgia							
Atlanta	110200038G02	1976	133	81	72	48	52
	"	1978	60	52	52	44	41[b]
	"	1980	106	73	64	42	41
	110200001P01	1976	123	95	94	56	61
	"	1978	73	69	69	44	45[b]
	"	1980	-	-	-	-	-
	110200039G01	1976	120	115	92	64	59
	"	1978	99	88	88	54	59[b]
	"	1980	98	93	72	42	39
	110200041G01	1976	100	92	83	45	51
	"	1978	74	60	60	32	33[b]
	"	1980	-	-	-	-	-
Macon	113440008F01	1976	109	85	71	36	40
	113440007F02	1978	84	58	53	30	33[b]
	113440007F02	1980	92	86	58	32	36

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations 10%	50%	Annual arithmetic mean ($\mu g/m^3$)
Idaho							
Boise City	130220007F01	1976	96	83	66	47	50
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Illinois							
Chicago	141220002P01	1976	172	140	130	91	91
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	141220001P01	1976	117	113	99	70	73
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Peoria	146080001P01	1976	94	72	68	52	51
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Indiana							
Indianapolis	152040025H01	1976	308	132	86	50	56
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	152040015H01	1976	128	122	89	53	54
	"	1978	-	-	-	-	-
	"	1980	92	90	90	51	55[b]
Iowa							
Bellevue	280180002F01	1976	126	97	87	41	46
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Kentucky							
Ashland	180080003F01	1976	94	93	79	46	48
	"	1978	173	123	72	30	35[b]
	"	1980	92	87	65	42	41
	180080008F01	1976	93	89	76	43	47
	"	1978	84	79	76	38	41
	"	1980	-	-	-	-	-
Paducah	183180020F01	1976	90	82	71	40	44
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations		Annual arithmetic mean ($\mu g/m^3$)
					10%	50%	
Louisiana							
Baton Rouge	190280002F01	1976	102	88	75	48	51
	"	1978	89	88	68	47	49
	"	1980	119	93	71	52	54
Maine							
Bangor	200100001F01	1976	126	103	80	50	51
	"	1978	80	70	61	42	46[b]
	"	1980	-	-	-	-	-
Maryland							
Baltimore	210120018F01	1976	137	134	95	60	63
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	210120007H01	1976	90	88	80	57	57
	"	1978	103	94	93	60	62[b]
	"	1980	-	-	-	-	-
Silver Spring	211480005G01	1976	98	82	66	38	39
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Michigan							
Detroit	231180001P01	1976	138	122	91	62	66
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	231180018F01	1976	123	115	105	62	68
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	231180016F01	1976	99	88	67	45	48
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Minnesota							
St. Paul	243300031P01	1976	91	83	71	52	54
	"	1978	126	113	86	56	58
	"	1980	103	86	68	49	47

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations		Annual arithmetic mean ($\mu g/m^3$)
					10%	50%	
Missouri							
Kansas City	171800012P01	1976	147	147	69	49	50
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
St. Louis	264280072P01	1976	136	127	109	71	73
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
	264280001P01	1976	111	105	94	64	59
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Nebraska							
Lincoln	281560004G01	1976	112	91	70	45	46
	"	1978	120	59	21	11	14
	281560012G01	1980	86	86	69	48	47
New Hampshire							
Nashua	300480005F01	1976	151	116	76	46	54
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	
North Carolina							
Belmont	340300001F02	1976	107	103	96	67	73
	"	1978	-	-	-	-	-
	340300003F02	1980	84	57	57	28	31[b]
Charlotte	340700001G01	1976	84	80	67	46	48
	"	1978	112	103	88	54	57
	"	1980	154	132	91	54	58
Winston-Salem	344460002G02	1976	80	68	65	43	45
	"	1978	95	91	76	39	43
	"	1980	148	94	55	24	33

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations 10%	50%	Annual arithmetic mean, ($\mu g/m^3$)
Ohio							
Akron	360060006H01	1976	96	91	70	45	46
	"	1978	139	109	87	52	57
	"	1980	93	93	75	53	54
	360060004H01	1976	100	91	82	48	53
	"	1978	135	126	82	47	53
	"	1980	86	83	76	52	52
Campbell	360960001101	1976	128	125	92	60	63
	"	1978	110	100	79	51	54
	"	1980	103	90	84	45	51
Cincinnati	361220018H01	1976	158	139	106	61	70
	"	1978	105	100	105	95	93[b]
	"	1980	-	-	-	-	-
	361220019P01	1976	121	98	89	61	62
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Cleveland	361300033H01	1976	193	181	127	83	88
	"	1978	259	247	197	92	109[b]
	"	1980	136	126	112	71	68
	361300012H01	1976	189	175	127	87	87
	"	1978	222	207	135	92	99
	"	1980	132	121	115	79	78
Moraine	364550001G01	1976	126	91	84	52	53
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Toledo	366600007H01	1976	117	115	80	53	56
	"	1978	162	116	92	58	64
	"	1980	102	101	90	59	59
Oklahoma							
Tulsa	373000112F01	1976	193	157	119	68	74
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Oregon							
Portland	381460001P01	1976	102	98	90	53	57
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations		Annual arithmetic mean, ($\mu g/m^3$)
					10%	50%	
South Carolina							
Mount Pleasant	421700001F01	1976	118	74	37	16	20
	"	1978	43	34	29	14	16
	"	1980	63	40	32	9	15[b]
Spartanburg	422040001F01	1976	121	90	74	30	42
	"	1978	105	99	83	28	33
	"	1980	119	45	43	3	18[b]
Tennessee							
Chattanooga	440380025G01	1976	94	92	74	48	51
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Eastridge	440900001G01	1976	124	95	64	46	47
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Knoxville	441740005G01	1976	119	114	101	70	70
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Nashville	442540002G01	1976	145	115	97	64	69
	"	1978	125	124	108	58	65[b]
	"	1980	156	140	113	54	60[b]
Texas							
Austin	450220004F01	1976	117	71	47	27	30
	"	1978	94	86	86	44	46[b]
	"	1980	75	70	55	32	35
	450220012F01	1976	93	79	58	20	24
	"	1978	106	77	57	17	22
	"	1980	-	-	-	-	-
Dallas	451310023H01	1976	91	88	77	50	51
	"	1978	231	190	112	54	63
	"	1980	-	-	-	-	-
	451310002F01	1976	97	96	83	52	52
	"	1978	238	165	127	74	77
	"	1980	265	202	183	84	90[b]
	451310002H01	1976	108	105	80	57	57
	"	1978	133	131	104	62	63
	"	1980	-	-	-	-	-

TABLE 8-10. (continued)

Location	Site Code	Year	Maximum ($\mu g/m^3$)	Second highest ($\mu g/m^3$)	Concentrations ($\mu g/m^3$) equalled or exceeded by stated percent of observations		Annual arithmetic mean ($\mu g/m^3$)
					10%	50%	
Fort Worth	451880021H02	1976	153	143	102	74	71
	"	1978	94	94	94	65	74[b]
	"	1980	-	-	-	-	-
	451880022H02	1976	138	124	95	63	61
	"	1978	91	66	91	61	59[b]
	"	1980	-	-	-	-	-
Houston	452560009H01	1976	162	137	127	56	64
	"	1978	170	116	102	66	64
	"	1980	128	101	99	30	46[b]
Utah							
Salt Lake City	460920001P01	1976	364	182	120	57	70
	"	1978	-	-	-	-	-
	"	1980	-	-	-	-	-
Washington							
Seattle	491840001P01	1976	119	114	91	66	65
	"	1978	139	113	113	90	93[b]
	"	1980	-	-	-	-	-
Wisconsin							
Milwaukee	512200045F01	1976	148	115	88	62	60
	"	1978	133	125	95	65	67
	"	1980	81	76	76	54	57[b]

[a] For comparison purposes, note that: 1.0 ppm NO_2 ≅ 1880 $\mu g/m^3$; 0.5 ppm ≅ 940 $\mu g/m^3$; 0.1 ppm ≅ 188 $\mu g/m^3$; 0.05 ppm ≅ 94 $\mu g/m^3$; and 0.01 ppm ≅ 18.8 $\mu g/m^3$.

[b] Data do not satisfy NADB minimum sampling criteria.

Since the National Ambient Air Quality Standard for NO_2 is stated in terms of the annual arithmetic mean, much attention has focused on long-term averages. One question which arises, then, is whether observed annual averages are an adequate index of the frequency and levels of short-term exposures. The data in Table 8-7, taken from SAROAD, show the ratios of the maximum hourly NO_2 concentrations to the annual means ov various cities for 1975. These ratios ranged from about 3.6 in Chicago, Illinois, to 13.5 in Dallas, Texas. Thus, it may be seen that these ratios, between the highest 1-hr NO_2 value during 1975 and the annual arithmetic mean for 1975, were quite different in various parts of the nation. To further illustrate this point, Figure 8-12 shows the distribution of maximum-to-mean NO_2 ratios averaged over the years 1972, 1973, and 1974 for 120 urban sites (Trijonis, 1978). Over 70 percent of the sites have maximum-to-mean ratios in the range of 5 to 8; about 8 percent of the sites have ratios exceeding 10. Figure 8-13 shows long-term trends in the maximum-to-mean NO_2 ratios for groups of sites in New Jersey and in the Los Angeles basin (Trijonis, 1978). (No Jacobs-Hochheiser data were used.) It is important to note that although the averaging procedure might be expected to smooth out fluctuations in the data, there is, nevertheless, no consistent value over the years for the ratio in either area. Looking back on Figures 8-4 to 8-8, marked variations in peak to mean ratios over many years at the same or different sites are apparent. It may be concluded, therefore, that the annual mean is not a good indicator of the highest short-term exposure level in the geographic areas considered.

Table 8-8 shows the frequency distribution of 1-hr NO_2 measurements at various sites in 1975 (U.S. EPA, 1977a). It may be seen here that there was great variability across the nation for all the percentile values presented. Also, it is obvious that the median value is not always indicative of the potential for short-term exposure. For instance, Los Angeles, Riverside, and San Diego, California, had high median values of 94 (0.05 ppm), 113 (0.06 ppm), and 113 $\mu g/m^3$ (0.06 ppm) respectively. St. Louis, Missouri, on the other hand, had the moderate median value of 75 $\mu g/m^3$ (0.040 ppm), but exceeded more than half the California sites reported for the one-percentile level. It may also be seen from this table that some small cities may experience peak concentrations of NO_2 even higher than those observed in center-city locations in major metropolitan areas. Ashland, Kentucky, reported a maximum of 895 $\mu g/m^3$ (0.48 ppm), which exceeded the maximum reported in most major metropolitan areas across the country. A similar conclusion may be drawn from Table 8-9, in that Port Huron, Michigan, reported a peak value of 832 $\mu g/m^3$ (0.44 ppm), which is exceeded in the listing only by Anaheim, California.

Two major factors that affect NO_2 concentrations, mobile source emissions and photochemical oxidation, have fairly consistent diurnal patterns in most urban areas. These usually contribute to the observed diurnal variation in NO_x concentrations. Such a variation is typified by a rapid increase in NO_2 in the morning as the result of NO emissions and photochemical conversion to NO_2. This is followed by a decrease of NO_2 in the midmorning hours due to advection and increasing vertical dispersion and also loss of NO_2 in various atmospheric

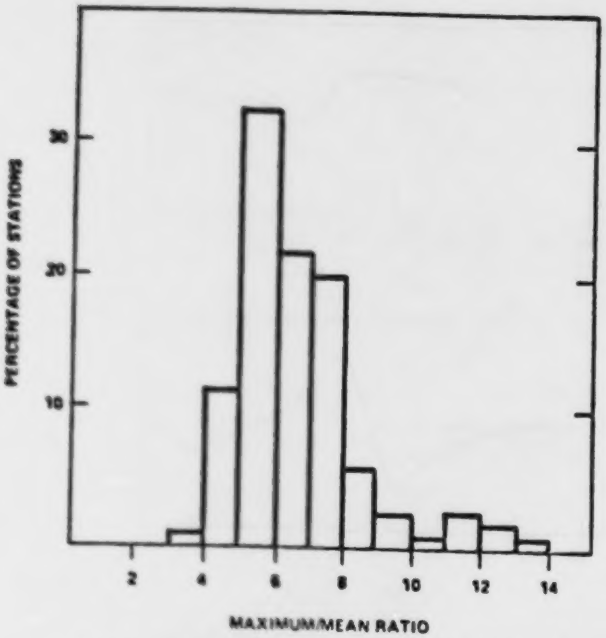

Figure 8-12. Distribution of maximum/mean NO_2 ratios for 120 urban locations averaged over the years 1972, 1973, and 1974 (adapted from Trijonis, 1978).

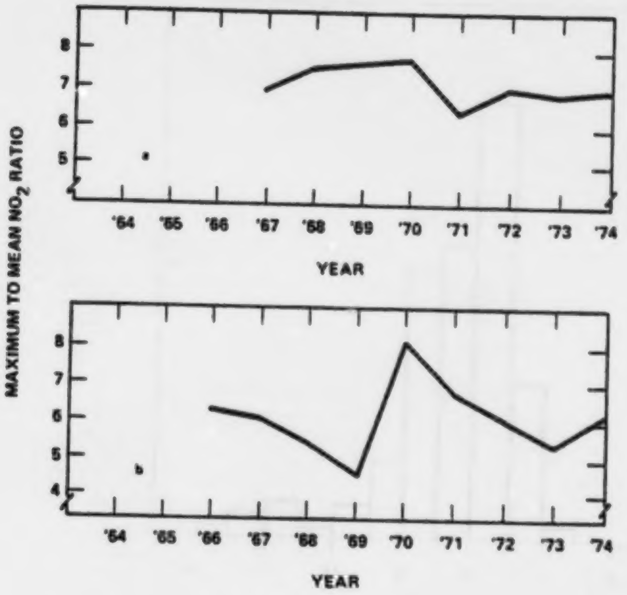

Figure 8-13. Trends in the maximum mean NO_2 ratio for two groups of sites: (a) average of five locations within the Los Angeles Basin (Anaheim, La Habra, Azusa, Pomona, San Bernadino); (b) average of two New Jersey sites (Bayonne and Newark).

Source: Adapted from Trijonis (1978).

transformation reactions. Peaks in the NO_2 concentration are often observed corresponding to emissions occurring during the late afternoon rush hour. In some areas, small lunchtime maxima occur. At many sites evening peaks occur. Ground-level NO concentrations usually build up slowly during the night.

To illustrate variations of NO_2, concentration data from the month of the highest observed 1-hr NO_2 concentration in three cities are presented in Figure 8-14 (U.S. EPA, 1975b). The monthly average 1-hr measurements were computed separately for each hour of the day. This gives the composite diurnal pattern as shown in Figure 8-14 for the month containing that year's highest reported short-term (1-hr) concentration. The data from Los Angeles during January 1975, and Denver during April 1975, followed the "typical" urban pattern described above, although the average NO_2 levels in Los Ageles were considerably higher. The pattern for Chicago during June 1975, was quite different. The extremely broad peak with the maximum between 2:00 and 3:00 p.m. was the result of individual daily maxima which did not occur at the same time of day during the month of June. This is one illustration of the fact that no standard diurnal pattern exists nationwide.

To further illustrate the diurnal trends in the same three cities, data from the day of the highest 1-hr NO_2 concentration in 1975 are plotted (U.S. EPA, 1975b) (Figure 8-15). In all three cities the diurnal patterns are similar to the average patterns. The extremely sharp, high peak in the Los Angeles data exemplifies the combined effects of poor atmospheric dispersion, high emissions, and photochemical activity which are quite common in this region.

In Figure 8-16, 1-hr average NO_2 concentration data are plotted versus time for periods of 3 days during which high NO_2 levels were observed (U.S. EPA, 1975b). The Los Angeles data showed a diurnal profile typical for the area on the afternoon on January 15, during which the NO_2 concentration climbed steadily after a small morning peak. This situation was probably the result of low wind speeds and a strong elevated inversion restricting both advection and dilution. The data for Ashland, Kentucky, show the same basic diurnal trend seen in Los Angeles on the first two days and much lower levels on the third day. The data from McLean, Virginia, at much lower overall concentration values, had quite a different pattern. The major increase in NO_2 concentration did not take place until 5:00 or 6:00 p.m.

Figure 8-17 shows the NO_2 and NO concentration profiles obtained from a center-city station in St. Louis, Missouri, and the NO_2 concentration from a rural site, 45 km north of the center-city location.[12] The center-city site showed a rapid buildup of NO during the morning with a slower rise in the NO_2 concentration. The rural site, at this time, reported NO_2 concentrations at or near the instrumental limits of detection. During the morning and early afternoon, the winds experienced at both sites were light and from the northwest. Between 1:00 and 2:00 p.m. the wind direction at the center-city site shifted and began coming from the south and south-east over the next several hours. A similar change occurred between 5:00 and 6:00 p.m. at the rural site. After the shift in direction, the wind speed increased somewhat at the rural site, and more gradually increased in the downtown area. Since other

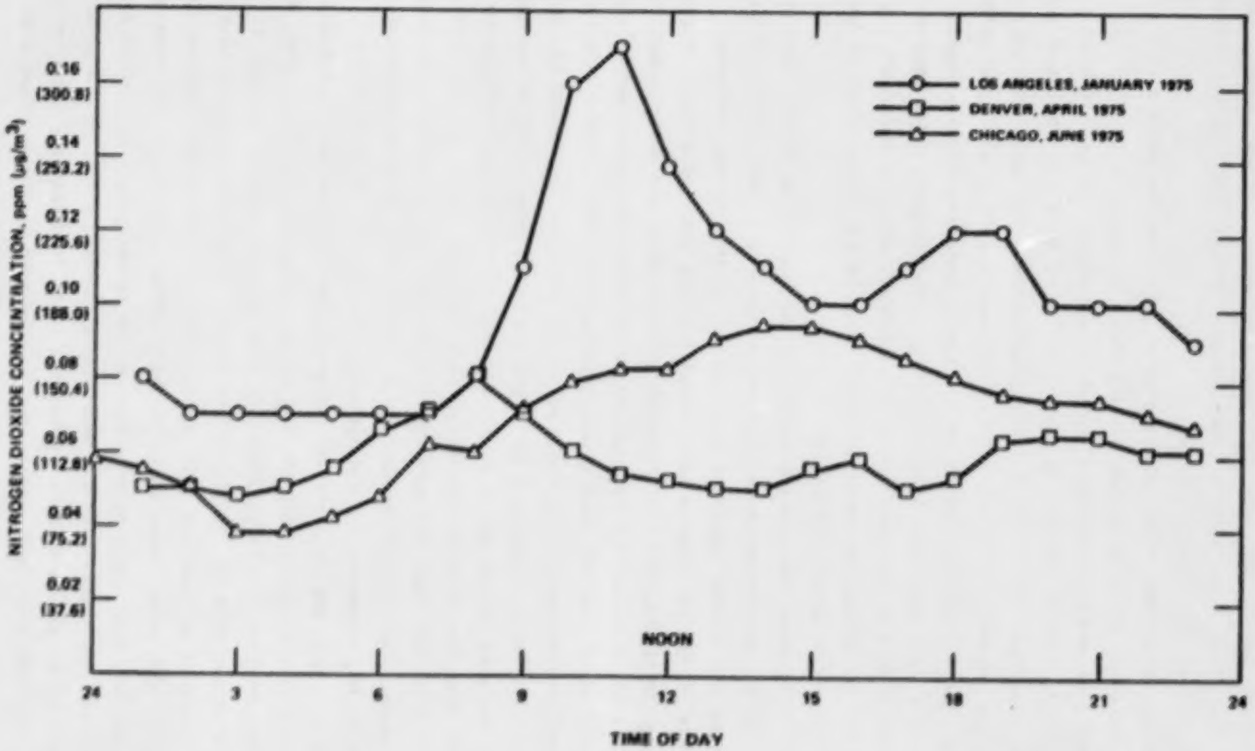

Figure 8-14. Average diurnal pattern for the month during which the highest 1-hour NO_2 concentrations were reported (U.S. EPA, 1975b).

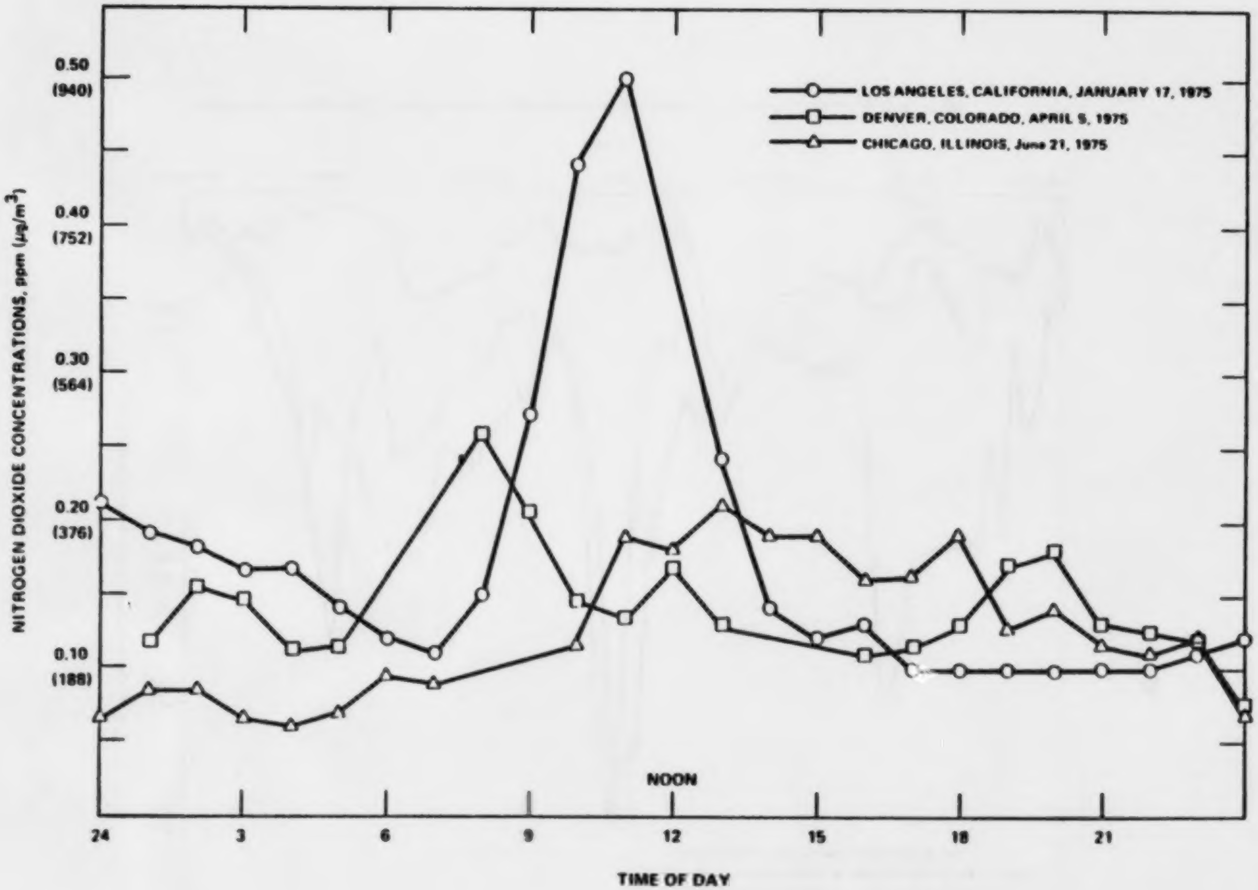

Figure 8-15. One-hour average concentration profiles on day of peak NO_2 concentration for three U.S. cities (U.S. EPA, 1975b).

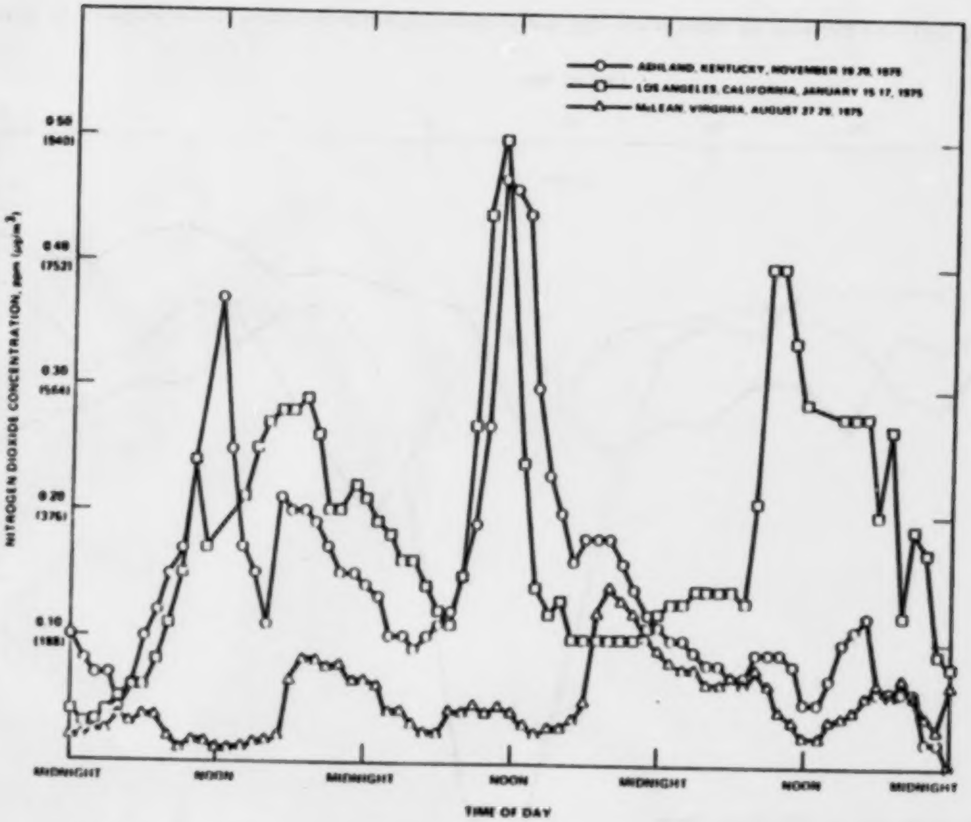

Figure 8-16. One hour NO_2 concentrations during three days of high pollution in three U.S. cities (U.S. EPA, 1975b).

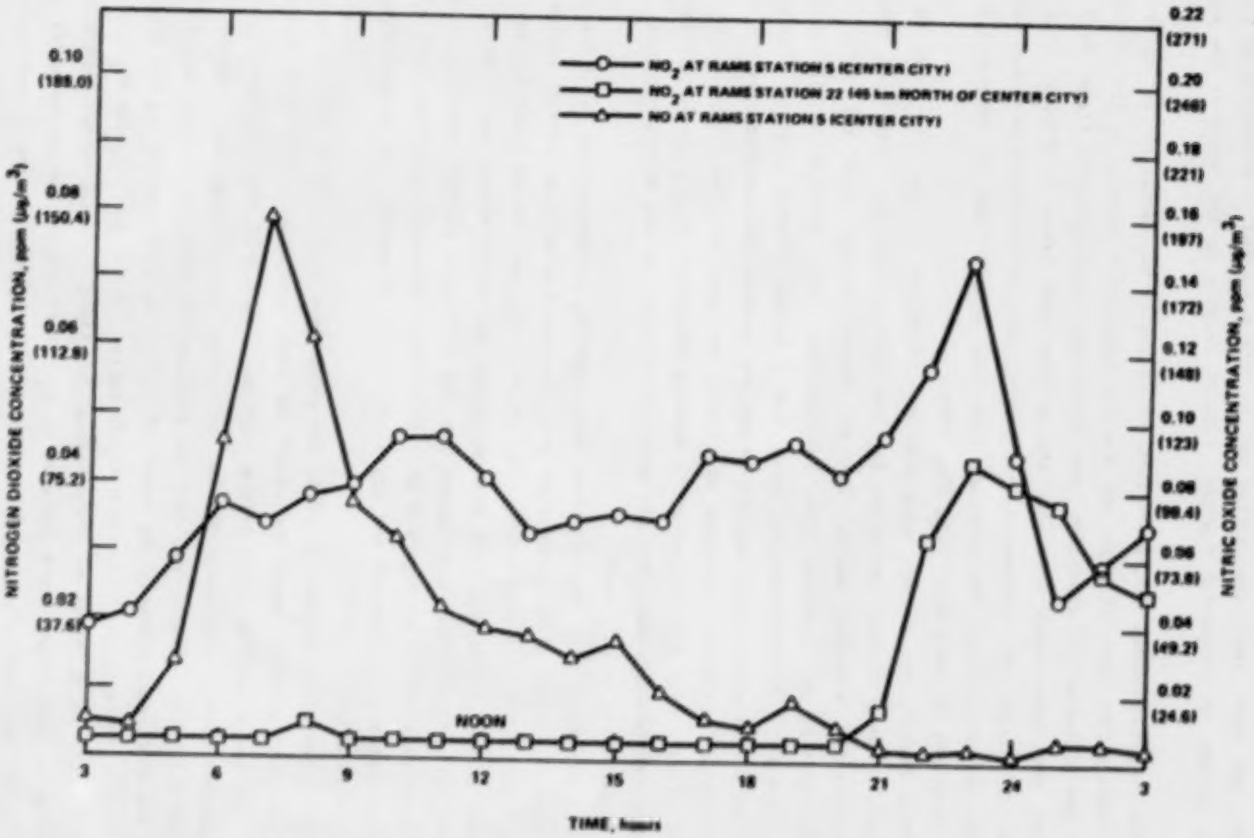

Figure 8-17. Nitric oxide and nitrogen dioxide concentrations at an urban and a rural site in St. Louis, Missouri, on January 27-28, 1976 (U.S. EPA, 1976b).

monitoring sites in and around St. Louis did not show a consistent, concomitant variation of NO_2 concentrations, the most likely explanation for the data presented is dispersion or plume impaction from a variety of industrial sources, located roughly to the south of the sites reported herein.

The above discussion indicates that short-term excursions of NO_2 concentrations to levels well above the average can occur at night and are not necessarily associated directly with traffic emission and photochemical oxidation, even though the levels shown in Figure 8-17 are considerably lower than those associated with the morning peaks shown in Figures 8-15 and 8-16. To further illustrate this phenomenon, the monthly maximum concentrations observed at each hour of the day are listed for selected individual months from six geographically-dispersed urban locations in Table 8-11 (U.S. EPA, 1975b).

The data from Newark, New Jersey, show peak concentrations in the late hours before midnight, and only a mild diurnal variation for the rest of the day. Portland, Oregon, experienced consistently elevated maxima for all hours between 2:00 and 11:00 p.m. Los Angeles, California, as expected from the previous data, experienced the highest short-term NO_2 levels in the mid-morning hours and a marked diurnal pattern. Chicago, Illinois, exhibited only a mild diurnal pattern in monthly maximum concentration for the month illustrated. In Denver, Colorado, elevated NO_2 levels are apparent from 9:00 a.m. to 7:00 p.m. In El Paso, Texas, both morning and early evening elevations in NO_2 concentrations are apparent. It also can be seen that, for all hours on at least one day during the month, NO_2 concentrations exceeded the monthly mean.

To summarize, the above data indicate that very high NO_2 concentrations, of a few hours duration, can occur in urban areas associated with the Los Angeles-type diurnal pattern of photochemical air pollution. In some cities, the diurnal NO_2 peak can be lower but may last longer. In other areas, relatively high concentrations may occur almost any time of day. Some qualitative insight into possible causes for the different diurnal patterns observed may be gleaned from examination of Figure 8-18. This figure presents data from RAPS on NO, NO_2, and O_3 concentrations for part of one day in St. Louis, Missouri. It is presented for illustrative purposes only.

On the day in question, October 1, 1976, the winds were sufficiently calm so that the pollutant profiles and patterns were the result of local processes rather than pollutant transport. In the early morning hours before sunrise, Figure 8-18 shows a significant and constant NO_2 concentration, presumably carried over from the previous day. During these hours, the O_3 concentration is quite low, near the instrumental detection limits. Nitric oxide concentrations are high, most probably the result of continuous emissions during the night and early morning hours. There is no discernible NO_2 formation. After sunrise, the NO_2 concentration increased sharply as a result of photochemical reactions. Photochemical generation of NO_2 is followed by a concomitant rapid increase in O_3 concentrations, which depresses the NO concentrations until the later afternoon hours when decreasing radiant energy and increasing

TABLE 8-11. DISTRIBUTION BY TIME OF DAY OF ONE-HOUR MAXIMUM NO_2 CONCENTRATIONS[a] FOR ONE MONTH IN 1975 FOR SELECTED URBAN SITES (U.S. EPA, 1976b)
($\mu g/m^3$)

	Newark New Jersey (July)	Los Angeles California (September)	Denver Colorado (November)	Portland Oregon (May)	Chicago Illinois (September)	El Paso Texas (October)
12 am	160	320	---[b]	110	180	94
1 am	160	280	130	94	160	75
2 am	160	240	130	94	160	75
3 am	160	210	110	94	150	75
4 am	140	240	160	94	150	75
5 am	140	240	190	75	160	170
6 am	160	260	230	75	220	75
7 am	180	430	240	94	260	150
8 am	200	850	170	110	210	150
9 am	210	1100	300	110	220	150
10 am	230	660	300	110	240	130
11 am	200	260	320	130	300	110
12 am	200	240	330	110	340	56
1 pm	180	230	250	110	260	94
2 pm	200	190	210	170	260	94
3 pm	200	170	300	170	260	75
4 pm	200	170	230	150	250	170
5 pm	230	230	250	150	230	150
6 pm	200	240	240	150	210	230
7 pm	160	260	300	150	210	210
8 pm	180	260	270	150	200	170
9 pm	230	280	220	150	200	130
10 pm	350	300	180	150	180	110
11 pm	330	300	140	130	200	110
Monthly average of all hours	96	130	98	51	110	51

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[a] Data presented to two significant figures only.

[b] No data available.

NO emissions overwhelm the ozone generating mechanism. From about 4:00 to 6:00 p.m. there is still sufficient ozone present to oxidize NO to NO_2 rapidly in a simple titration reaction apparently not involving hydrocarbons. This ozone scavenging mechanism would seem to be identical to that observed in plumes from power plants (Section 6.1.3). Significant quantities of NO are oxidized leading to high nighttime NO_2 concentrations. It may be postulated that this mechanism is operative also in other localities exhibiting elevated NO_2 levels after photochemical activity, including NO_2 photodissociation, decreases in the evening. Rapid reaction of NO and O_3 leading to increased NO_2 concentrations has also been observed across a high-traffic-volume freeway in Los Angeles (Fankhauser, 1977).

Although the data presented in Figure 8-18 were carefully chosen as an unusually good example of ozone scavenging, Table 8-12 shows that elevated NO_2 levels in the late evening hours are a fairly common phenomenon in the Greater St. Louis area. Fifty-three of the 89 high NO_2 values reported in Table 8-12 occurred between the hours of 7:00 p.m. and 6:00 a.m.

Variations in the values for peak concentrations and annual means from station to station for the densely-monitored St. Louis area documented in Table 8-12 are also an indication of the possible importance of local sources and small-scale meteorological and topographical features in determining ambient pollutant concentrations.

8.1.7 Spatial and Temporal Variations of NO_2 Concentrations as Related to Estimation of Human Exposure

Currently, if a single monitoring station in an Air Quality Control Region (AQCR) reports ambient concentrations in excess of that safe for human health and welfare, this fact is considered cause for concern. While this assumption appears reasonable if human health and welfare are to be protected, particularly if the monitoring station has been located in urban or other population centers specifically to monitor population exposure (SAROAD purpose designation 01, population-oriented), it may be of interest for some purposes to consider methods for estimating human exposures in more detail. Some considerations involved in making such exposure estimates are discussed in this section.

8.1.7.1 Spatial and temporal variations of local NO_2 concentrations--Table 8-13 gives the NO_2 hourly concentrations measured at all 25 RAMS sites in St. Louis during a period of high pollution for this area from 7 a.m. on October 1, 1976 to 2 a.m. on the following day. It is presented as one illustration of the geographic and temporal variability of maximum NO_2 concentrations which may be experienced in a single airshed. It should be noted that few areas in this country are as densely monitored as St. Louis was during 1975-76.

Sources of NO_x in the St. Louis area include mobile vehicles, other area sources, and a wide variety of industrial point sources. The data presented are not necessarily to be taken as typical of urban areas since the impact of a number of factors including source emission strengths, their diurnal emission patterns, meteorological factors, topography and the presence of other photochemical pollutants and their precursors may vary from area to area. In general, a meaningful survey of the phenomenology or possible causes of the variability of high NO_2 concentrations on a nationwide basis is lacking in the literature.

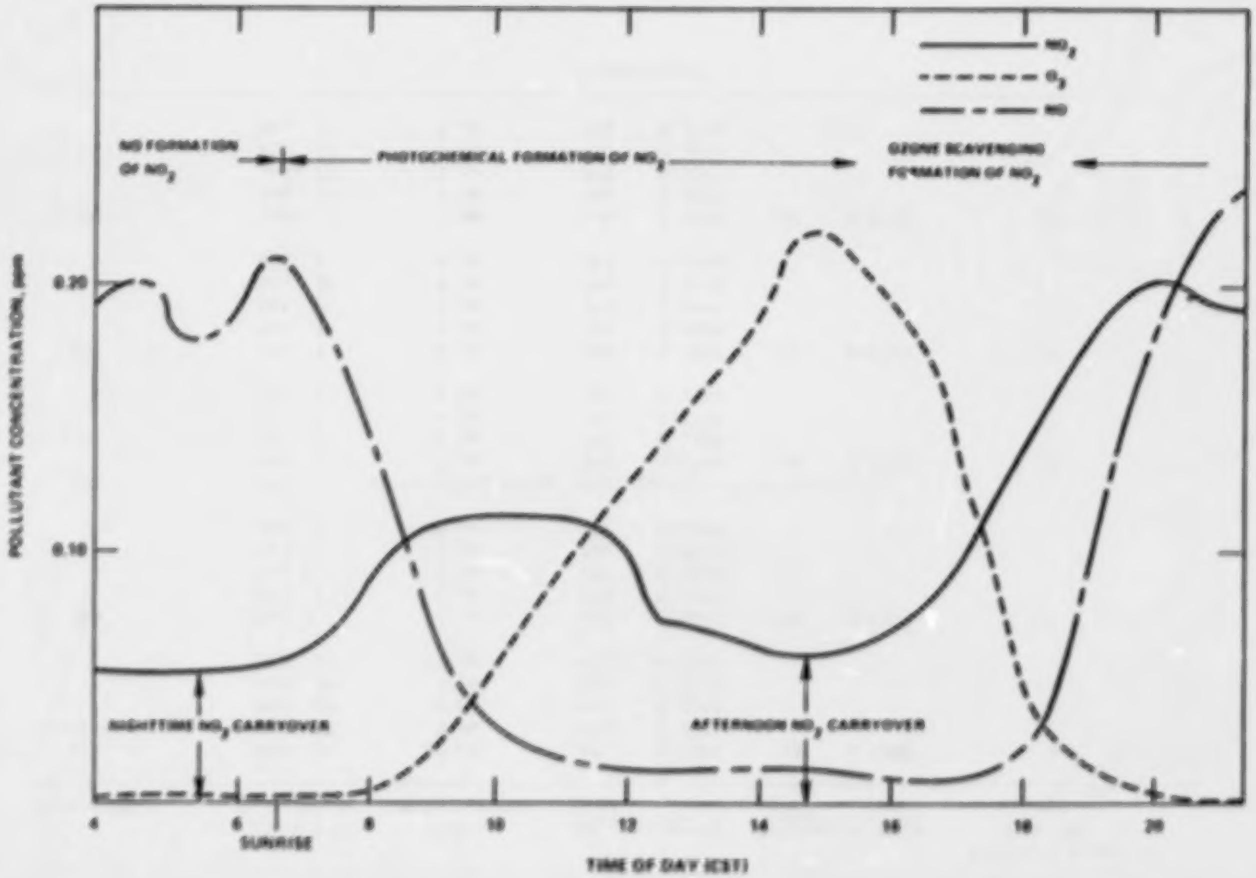


Figure 8-18. Pollutant concentrations in Central City St. Louis, October 1, 1976, average of RAMS sites 101, 102, 106, and 107. Illustration of photochemical and ozone scavenging formation of NO_2 (U.S. EPA, 1976b).

TABLE 8-12. MEAN AND TOP FIVE HOURLY NITROGEN DIOXIDE CONCENTRATIONS REPORTED FROM 18 INDIVIDUAL RAMS STATIONS IN ST. LOUIS DURING 1976 (U.S. EPA, 1976b)

Site Number	Date of Measurement	Time of Measurement	Concentration $\mu g/m^3$	Concentration ppm	Arithmetic Mean $\mu g/m^3$	Arithmetic Mean ppm	Distance from Site 101 (km)
101 (Center-city)	Nov. 19	10 pm	481	0.2556	53	0.0282	0
	Nov. 19	9 pm	454	0.2415			
	Nov. 19	8 pm	443	0.2358			
	Nov. 19	7 pm	434	0.2310			
	Nov. 19	11 pm	411	0.2187			
104	May 1	7 am	293	0.1559	48	0.0253	≤4
	Oct. 1	6 pm	291	0.1549			
	May 1	8 am	287	0.1526			
	Oct. 1	5 pm	284	0.1512			
	Oct. 2	6 pm	284	0.1509			
105	----------------first value invalidated----------------						
	Oct. 1	7 pm	350	0.1864	44	0.0235	≤4
	Oct. 1	10 pm	337	0.1795			
	Oct. 1	8 pm	334	0.1776			
	Oct. 1	9 pm	332	0.1768			
107	Oct. 1	6 pm	358	0.1907	57	0.0305	≤4
	Oct. 1	7 pm	353	0.1880			
	Oct. 2	7 pm	271	0.1441			
	Aug. 25	9 am	266	0.1413			
	Oct. 2	8 am	263	0.1400			
108	Sept. 1	7 am	636	0.3383	33	0.0174	≤20
	Aug. 30	8 am	566	0.3010			
	July 26	11 am	321	0.1707			
	Aug. 30	9 am	312	0.1659			
	Oct. 2	8 am	291	0.1548			

(continued)

TABLE 8-12. (continued)

Site Number	Date of Measurement	Time of Measurement	Concentration $\mu g/m^3$	Concentration ppm	Arithmetic Mean $\mu g/m^3$	Arithmetic Mean ppm	Distance from Site 101 (km)
109	Apr. 30	8 am	289	0.1537	26	0.0138	≤20
	Oct. 1	6 pm	216	0.1147			
	Oct. 1	7 pm	197	0.1049			
	Oct. 1	8 pm	186	0.0991			
	Apr. 7	12 am	173	0.0922			
114	Oct. 2	8 am	305	0.1524	32	0.0172	≤20
	Oct. 2	12 am	281	0.1495			
	Oct. 2	3 am	276	0.1466			
	Oct. 1	9 pm	275	0.1462			
	Oct. 2	9 am	273	0.1452			
115	Dec. 4	10 am	286	0.1520	22	0.0116	≤20
	Oct. 2	8 am	170	0.0903			
	May 10	10 am	152	0.0809			
	Sept. 22	9 am	145	0.0771			
	July 6	11 pm	128	0.0682			
116	May 7	11 pm	457	0.2430	23	0.0120	≤20
	May 7	10 pm	326	0.1734			
	Oct. 2	7 pm	228	0.1214			
	Oct. 1	8 pm	206	0.1098			
	Oct. 1	7 pm	206	0.1094			
102	Oct. 1	7 pm	374	0.1990	62	0.0329	≤10
	Oct. 2	8 am	365	0.1940			
	Oct. 2	9 am	363	0.1929			
	Oct. 1	6 pm	354	0.1882			
	Oct. 1	8 pm	331	0.1762			

(continued)

TABLE 8-12. (continued)

Site Number	Date of Measurement	Time of Measurement	Concentration $\mu g/m^3$	Concentration ppm	Arithmetic Mean $\mu g/m^3$	Arithmetic Mean ppm	Distance from Site 101 (km)
106	Oct. 1	10 pm	460	0.2449	56	0.0298	≤ 10
	Oct. 1	9 pm	446	0.2375			
	Oct. 1	11 pm	442	0.2352			
	Oct. 1	7 pm	408	0.2169			
	Oct. 1	8 pm	405	0.2156			
110	Feb. 24	7 am	405	0.2155	34	0.0182	≤ 10
	Oct. 1	7 pm	263	0.1398			
	Oct. 1	6 pm	257	0.1366			
	Oct. 1	10 pm	201	0.1069			
	Oct. 1	11 pm	200	0.1067			
111	Oct. 1	8 pm	419	0.2230	45	0.0241	≤ 10
	Oct. 1	7 pm	406	0.2161			
	Oct. 1	9 pm	399	0.2121			
	Oct. 1	6 pm	387	0.2060			
	Oct. 1	10 pm	368	0.1956			
112	Oct. 1	11 pm	318	0.1689	52	0.0275	≤ 10
	Oct. 1	10 pm	317	0.1686			
	Aug. 23	8 am	315	0.1677			
	Oct. 1	7 pm	308	0.1637			
	Oct. 1	9 pm	308	0.1636			
117	May 19	2 am	676	0.3594	21	0.0110	≤ 20
	May 19	6 am	566	0.3008			
	May 19	4 am	544	0.2891			
	May 19	3 am	461	0.2450			
	May 19	5 am	360	0.1914			

(continued)

TABLE 8-12. (continued)

Site Number	Date of Measurement	Time of Measurement	Concentration $\mu g/m^3$	Concentration ppm	Arithmetic Mean $\mu g/m^3$	Arithmetic Mean ppm	Distance from Site 101 (km)
118	Sept. 17	8 am	149	0.0791	21	0.0111	≤20
	Sept. 15	10 am	136	0.0722			
	Nov. 6	9 pm	134	0.0715			
	Nov. 6	8 pm	132	0.0705			
	Nov. 6	7 pm	127	0.0677			
119	Feb. 3	11 pm	360	0.1917	35	0.0184	µ20
	Feb. 3	10 pm	343	0.1825			
	Feb. 3	8 pm	336	0.1787			
	Feb. 3	9 pm	336	0.1786			
	Feb. 3	7 pm	316	0.1681			
120	Oct. 11	6 pm	360	0.1916	37	0.0198	≤20
	Sept. 3	7 am	296	0.1430			
	Oct. 11	8 am	258	0.1372			
	Oct. 11	7 pm	248	0.1322			
	Aug. 23	8 am	236	0.1254			

Table 8-13. GEOGRAPHICAL VARIATION OF HOURLY NO_2 CONCENTRATIONS DURING A PERIOD OF HIGH NO_2 CONCENTRATIONS (U.S. EPA, 1976b)
[St. Louis, October 1 and 2, 1976 (ppm)]

RAMS	Hour of Day																				
Station		7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	1	2
101	0.07	0.12	0.12	0.18	0.14	0.08	0.09	0.09	0.10	0.06	0.12	0.19	0.20	0.19	*	0.18	0.16	0.17	*	*	
102	0.07	0.12	0.17	0.11	0.12	0.10	0.08	0.06	0.05	0.07	0.13	0.19	0.20	0.18	0.17	*	0.13	*	*	*	
103	0.05	0.10	0.15	0.12	0.10	0.07	0.06	0.04	0.02	0.03	0.06	0.15	0.16	0.13	0.14	0.15	0.13	0.12	0.09	0.08	
104	*	*	*	*	*	*	0.07	0.07	0.07	0.08	0.15	0.15	0.15	0.15	0.13	0.13	0.11	0.09	0.08	0.07	
105	0.05	0.07	0.12	0.14	0.07	0.04	0.05	0.07	0.10	0.06	0.08	0.17	0.19	0.18	0.18	0.18	0.13	0.11	0.08	0.08	
106	0.07	0.11	0.10	0.08	0.08	0.06	0.06	0.05	0.06	0.10	0.11	0.17	0.22	0.22	0.24	0.24	0.24	0.20	0.17	0.15	
107	0.07	0.08	0.08	0.08	0.11	0.09	0.07	0.05	0.07	0.08	0.14	0.19	0.19	*	*	*	*	*	*	0.12	
108	0.06	0.10	0.09	0.08	0.05	0.05	0.02	0.03	0.02	0.02	0.04	0.10	0.04	0.09	0.11	0.12	0.11	0.11	0.09	0.10	
109	*	*	*	*	0.02	0.01	0.02	0.03	0.02	0.02	0.04	0.11	0.10	0.10	0.08	0.08	0.07	0.05	0.04	0.04	
110	0.03	0.04	0.08	0.07	0.04	0.01	0.02	0.04	0.06	0.05	0.08	0.14	0.14	0.10	0.10	0.11	0.11	0.09	0.07	0.07	
111	0.05	0.07	0.08	0.06	0.04	0.04	0.03	0.03	0.04	0.06	0.11	0.21	0.22	0.22	0.21	0.20	0.17	0.13	0.12	0.08	
112	0.05	0.06	0.05	0.06	0.08	0.05	0.04	0.02	0.03	0.06	0.16	0.15	0.16	0.16	0.16	0.17	0.17	0.15	0.15	0.13	
113	*	*	*	*	*	*	*	*	*	*	*	*	*	*	0.21	0.20	0.21	0.21	0.18	0.17	

(continued)

TABLE 8-13. (continued)

RAMS Station	Hour of Day																			
		7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	1	2
114	*	*	*	*	0.04	0.03	0.03	0.03	0.01	0.01	0.04	0.07	0.07	0.09	0.15	0.13	0.13	0.15	0.14	0.14	
115	0.04	0.07	0.07	0.05	0.03	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.05	0.03	0.03	0.03	0.06	0.05	0.04	0.03	
116	0.04	0.06	0.04	0.03	0.02	0.01	0.01	0.01	0.02	0.02	0.03	0.05	0.11	0.11	0.10	0.07	0.05	0.04	0.04	0.05	
117	0.03	0.02	0.02	0.01	0.01	0.01	0.00	0.01	0.02	0.02	0.02	0.02	0.02	0.04	0.07	0.07	0.06	0.04	0.07	0.05	
118	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.02	0.02	0.03	0.04	0.03	0.03	0.03	0.05	0.06	0.04	0.03	0.06	0.05	
119	0.04	0.04	0.04	0.03	0.03	0.02	0.02	0.02	0.02	0.03	0.04	0.07	0.10	0.12	0.11	0.10	0.10	0.10	0.09	0.07	
120	*	*	*	*	*	*	*	0.01	*	*	*	*	*	*	*	*	*	*	*	*	
121	0.05	0.04	0.02	0.01	0.03	0.02	0.01	0.00	0.00	0.01	0.01	0.02	0.08	0.14	0.16	0.13	0.12	0.17	0.14	0.09	
122	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.02	*	*	*	
123	0.00	*	*	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	*	*	*	*	*	*	*	*	
124	0.03	0.02	0.01	0.01	0.01	0.01	0.00	0.00	0.02	0.03	0.04	0.03	0.02	0.01	0.01	0.01	0.01	0.02	0.02	0.01	
125	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	

*Data missing.

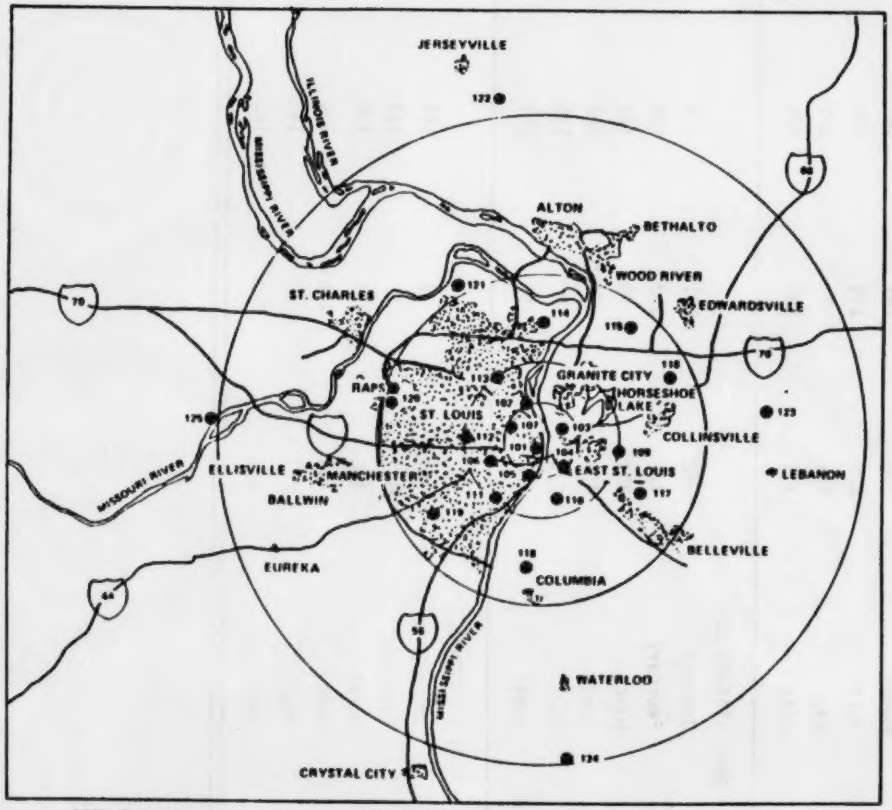
Figure 8-19 shows the locations of the 25 RAMS sites. The concentric circles locate these stations within distances of 4, 10, 20 and 40 km of "center-city" Station 101. Comparison of Table 8-14 and Figure 8-19 reveals that with a 4 km radius the peak hourly NO_2 concentrations varied from 0.15 to 0.20 ppm; within a 10 km radius values ranged from 0.15 to 0.24 ppm; within a 20 km radius a four-fold variation from 0.06 to 0.24 was experienced; and within a 40 km radius values ranging from 0.01 to 0.24 ppm were reported. The major peak occurred after sundown at almost all sites, presumably due to the ozone titration mechanism. A subsidiary photochemical peak is discernible in the late morning at central sites. Significant NO_2 carry-over is evident at the start of the period reported. Both the duration and time of occurrence of peak hourly values is variable from station to station as indicated by the underlined portions in Table 8-13.

Another way of viewing the extent of the variability of NO_x concentrations in the St. Louis area is presented in Table 8-14. This table is intended to illustrate the occurrence of high NO_2 concentrations shown by monitoring throughout an airshed even though the events may not take place at the same times and may not be associated with the same air mass. The sites represented in the tables are located in a center-city location subject to nearby automobile and truck traffic (101); in an outlying city location within a few kilometers of an electric power plant and a number of heavy industrial sites (104); and in a high-density single family residential community (111). It can be seen from these tables that both the highest NO hourly readings and the corresponding NO/NO_2 ratios are quite different for Station 111 than for Stations 101 and 104. Some evidence of spatial smoothing in both the highest and second highest hourly NO_2 concentrations by month is apparent in the data.

A somewhat different scenario is presented by the same type of data (Fairfax County; Keyes et al.) (Tables 8-15 through 8-17) from three monitoring stations in Fairfax County, Virginia, near Washington, D.C. These stations are located in an urban complex dominated by area sources (70 percent of NO_x emissions from area sources). Locations for the monitoring stations cited in the table include an office complex, a high-volume transportation intersection, and a suburban commercial center. The maximum separation between stations is approximately 15 km.

The data reported show a considerable variation in the ratio of the highest hourly NO to the corresponding hourly NO_2, suggesting a considerable variation in local NO area emissions or in monitor siting. Nevertheless, it is important to note that the highest hourly NO_2 concentrations reported by month are quite similar at all stations. Presently, an insufficient number of analyses have been conducted on a nationwide basis to determine whether or not this observation is typical of area-source-dominated urban airsheds.

In a final scenario, very recent ground level NO_x measurements from a large isolated source in complex terrain are presented (Pickering, 1980). (Individual NO_2 values were not reported.) Measurements of this type are extremely rare in the literature. Exhaust gases from the 712 MW Clinch River power plant, burning low sulfur coal, are emitted through two


JERSEYVILLE
122
ILLINOIS RIVER
MISSISSIPPI RIVER
ALTON
BETHALTO
WOOD RIVER
EDWARDSVILLE
ST. CHARLES
70
121
114
115
113
116
GRANITE CITY
HORSESHOE LAKE
RAPS
120
102
ST. LOUIS
107
103
112
101
COLLINSVILLE
123
104
108
106
105
EAST ST. LOUIS
LEBANON
175
ELLISVILLE
MANCHESTER
BALLWIN
111
110
117
119
BELLEVILLE
MISSOURI RIVER
EUREKA
118
COLUMBIA
44
55
WATERLOO
124
CRYSTAL CITY
55

NOTE: Sites are aligned in approximate concentric circles of 4, 10, 20, and 40 km, respectively, adapted from Nelson, 1978

Figure 8 19. St. Louis RAMS station locations.

TABLE 8-14. HIGH CONCENTRATIONS OF NITROGEN OXIDES, ST. LOUIS, MISSOURI, 1976 (U.S. EPA, 1976b)

Month	Highest NO ($\mu g/m^3$)	Corresponding NO_2 ($\mu g/m^3$)	Highest NO_2 ($\mu g/m^3$)	2nd Highest NO_2 ($\mu g/m^3$)
RAMS STATION 101				
January	329	126	162	152
February	475	59	146	140
March	260	147	147	140
April	500	199	247	207
May	179	*	175	163
June	303	190	225	211
RAMS STATION 104				
January	362	111	111	96
February	474	103	194	173
March	303	99	121	120
April	406	91	293	293
May	276	*	155	152
June	228	99	151	126
RAMS STATION 111				
January	644	76	109	101
February	617	*	124	116
March	456	134	136	134
April	434	128	182	180
May	267	*	143	130
June	226	152	181	176

TABLE 8-15. MONTHLY TRENDS IN HOURLY NO AND NO_2 CONCENTRATIONS, MASSEY BUILDING STATION, FAIRFAX COUNTY, VIRGINIA, 1977[a] (KEYES ET AL.)

Month	Highest NO ($\mu g/m^3$)	Corresponding NO_2 ($\mu g/m^3$)	Highest NO_2 ($\mu g/m^3$)	2nd Highest NO_2 ($\mu g/m^3$)
January	655	140	160	150
February	650	105	190	170
March	350	105	140	130
April	230	120	160	
May	40	20	180	130
June	80	85	170	160
July	20	65	85	75
August	70		95	75
September	125	95	115	105
October	420	75	225	130
November	680	43	115	105
December	645	120	120	115

Annual Average NO_2: 40 $\mu g/m^3$

Second Highest NO_2: 190 $\mu g/m^3$

Peak/mean = 4.8

[a] Data from Fairfax County (Va.) Air Pollution Control Agency.

TABLE 8-16. MONTHLY TRENDS IN HOURLY NO AND NO_2 CONCENTRATIONS, LEWINSVILLE STATION, FAIRFAX COUNTY, VIRGINIA, 1977[a] (KEYES ET AL.)

Month	Highest NO ($\mu g/m^3$)	Corresponding NO_2 ($\mu g/m^3$)	Highest NO_2 ($\mu g/m^3$)	2nd Highest NO_2 ($\mu g/m^3$)
January	680	65	130	125
February	630	65	290	225
March				
April	615	95	190	180
May	380	120	290	255
June	290	180	225	205
July	290	75	170	140
August	650	10	280	265
September	515	75	280	235
October	580	40	160	150
November	700	45	140	130
December	680	75	130	120

Annual Average NO_2: 56 $\mu g/m^3$

Second Highest NO_2: 280 $\mu g/m^3$

Peak/mean = 5.0

[a] Data from Fairfax County (Va.) Air Pollution Control Agency.

TABLE 8-17. MONTHLY TRENDS IN HOURLY NO AND NO_2 CONCENTRATIONS, SEVEN CORNERS STATION, FAIRFAX COUNTY, VIRGINIA, 1977[a] (KEYES ET AL.)

Month	Highest NO ($\mu g/m^3$)	Corresponding NO_2 ($\mu g/m^3$)	Highest NO_2 ($\mu g/m^3$)	2nd Highest NO_2 ($\mu g/m^3$)
January	630	10	150	120
February	620	50	205	145
March	540	0	120	110
April	665	0	265	195
May	240	40	190	170
June	170	20	140	130
July	185	55	170	150
August	320	105	235	150
September	505	105	205	170
October	420	85	160	150

Annual Average NO_2: 46

Second Highest NO_2: 235

Peak/mean = 5.1

[a] Data from Fairfax County (Va.) Air Pollution Control Agency.

stacks, 42 m apart, each 138 m high. The surrounding terrain has ridges exceeding 1.5 times stack height within 3 to 5 km of the plant in all quadrants of the compass. Figure 8-20 shows the location and elevation of the monitoring sites. Table 8-18 gives the mean of one-hour NO_x concentrations over an extended monitoring period from November 20, 1976 to September 30, 1977. It is important to note that the average values reported are subject to large error since many readings were near the detection limit of the analytical instruments. The data do, however, indicate the magnitude of the average values. Much greater confidence can be placed in the individual 10 highest NO_x readings recorded at each site (Table 8-19). Perhaps the most significant point to note for this discussion are the extremely high peak to mean ratios for NO_x observed at all sites (compare Tables 8-18 and 8-19). Although detailed analysis of the frequency distribution of high NO_x concentrations has yet to be made, it seems appropriate to state that the probability is small for detecting the maximum impact of a plume with fixed monitoring stations. This observation may be expected to hold true also in regions subject to a variety of point and area sources. In terms of estimating human exposure due to large point sources, therefore, fixed monitoring sites may be expected to provide data mainly on the long-term average impact of such sources.

8.1.7.2 Estimating human exposure--The examples of local variability of significant NO_2 concentrations just presented have some bearing on the question of estimating human exposure to this pollutant. Such an estimation might include a study of the spatial variation of NO_2 concentrations for time periods during which human health and/or welfare are or may be adversely affected and might take into account the mobility of the population at risk. In an area like Fairfax County, it is possible that the relatively small spatial variation in high NO_2 concentrations which is evident from the data presented in Table 8-14 might enable estimates of exposure to high NO_2 concentrations to be made from data gathered at a single monitoring station. In an area like St. Louis, more detailed monitoring might be required due to the greater variation in ambient levels across this urban area. In addition, the occurrence of high NO_2 concentrations during the late evening hours when most of the population is presumably indoors would point to the desirability of considering relationships between indoor and outdoor pollutant concentrations. These are only a few examples of the methodological difficulties associated with detailed estimates of actual human exposure.

Few attempts to perform such exposure estimates have been reported in the literature. Recently, an exposure model has been reported for the Los Angeles area (Horie et al., 1977). The main thrust of this work was to estimate population exposure to NO_2 concentrations as a percentage of time the population was potentially exposed to NO_2 concentrations exceeding the California Ambient Air Quality Standard (CAAQS) (470 $\mu g/m^3$ or 0.25 ppm for 1-hr averages). Presentation of this model is in no sense intended to reflect on consideration of a National Ambient Air Quality Standard but is included only to illustrate an existing exposure methodology.

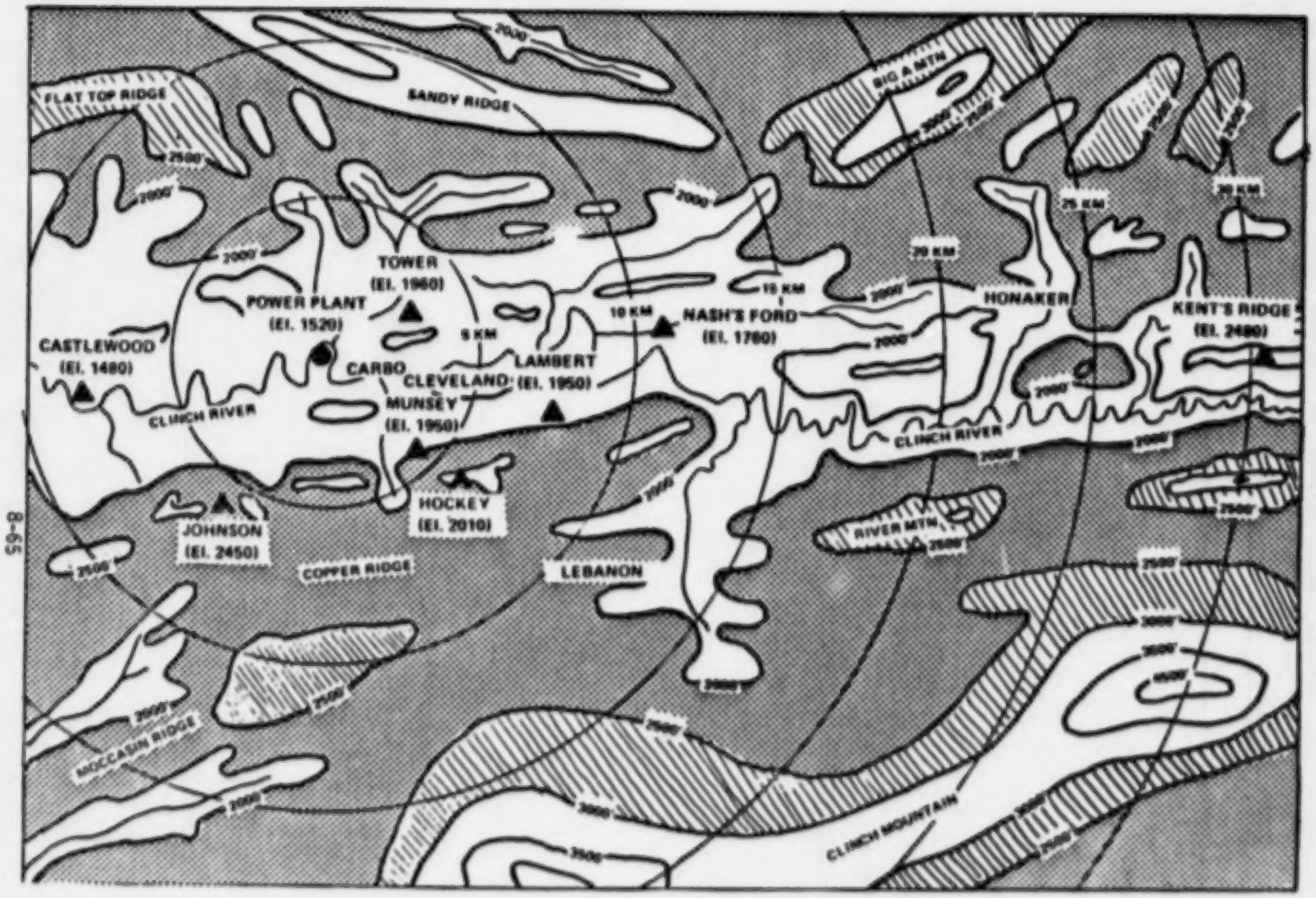

FLAT TOP RIDGE
SANDY RIDGE
BIG A MTN
2000'
2500'
25 KM
30 KM
20 KM
15 KM
10 KM
5 KM
TOWER
(El. 1960)
POWER PLANT
(El. 1520)
CASTLEWOOD
(El. 1480)
CARBO
CLEVELAND
LAMBERT
(El. 1950)
MUNSEY
(El. 1950)
NASH'S FORD
(El. 1780)
HONAKER
KENT'S RIDGE
(El. 2480)
CLINCH RIVER
HOCKEY
(El. 2010)
JOHNSON
(El. 2450)
COPPER RIDGE
LEBANON
RIVER MTN
MOCCASIN RIDGE
CLINCH MOUNTAIN
3000'
3500'

Figure 8-20. Location and elevation of Clinch River Power Plant monitoring stations (Pickering, 1980).

TABLE 8-18. MEAN NO_x CONCENTRATIONS FOR ISOLATED POINT SOURCE IN COMPLEX TERRAIN (CLINCH RIVER POWER PLANT) (ppb) (PICKERING, 1980)

Station	NO_x	NO_2	NO
Tower	29	15	15
Munsey	7	5	1
Hockey	5	3	2
Lambert	-	-	-
Johnson	-	-	-
Castle	25	11	13
Kents	11	7	3
Nashs	15	9	6

TABLE 8-19. TEN HIGHEST HOURLY AVERAGE NO_x CONCENTRATIONS OBSERVED AT EACH MONITORING SITE FOR ISOLATED SOURCE (CLINCH RIVER POWER PLANT) (PICKERING, 1980)

	Tower			Hockey			Kents		
Rank	NO_x Conc. (ppb)	Date	Hour	NO_x Conc. (ppb)	Date	Hour	NO_x Conc. (ppb)	Date	Hour
1	619	1/28/77	10	568	6/30/77	5	353	8/13/77	22
2	549	12/30/76	14	399	7/5/77	3	103	2/19/77	4
3	482	3/15/77	11	329	8/15/77	9	101	8/8/77	7
4	467	12/30/76	20	255	6/30/77	4	92	12/14/76	11
5	457	7/7/77	8	219	7/6/77	8	89	11/24/76	24
6	448	1/26/77	20	205	7/2/77	6	83	12/14/76	12
7	430	1/28/77	13	170	4/20/77	4	80	8/8/77	3
8	420	12/23/76	10	164	3/21/77	11	79	8/6/77	15
9	414	12/30/76	16	146	5/14/77	10	79	12/19/76	6
10	397	12/23/76	9	130	7/20/77	3	78	1/13/77	17

	Nashs			Castle			Munsey		
Rank	NO_x Conc. (ppb)	Date	Hour	NO_x Conc. (ppb)	Date	Hour	NO_x Conc. (ppb)	Date	Hour
1	834	8/10/77	16	816	2/15/77	15	419	3/9/77	9
2	601	6/7/77	12	589	2/8/77	11	408	3/9/77	8
3	553	8/10/77	15	464	2/8/77	10	297	7/6/77	9
4	262	2/10/77	12	435	2/11/77	9	280	7/6/77	8
5	235	7/8/77	10	410	2/11/77	10	276	3/9/77	7
6	229	2/10/77	11	394	2/11/77	11	252	3/9/77	10
7	228	7/12/77	10	375	2/8/77	12	206	6/16/77	8
8	226	2/11/77	12	365	2/11/77	12	186	3/21/77	11
9	211	6/7/77	13	350	2/10/77	10	173	3/9/77	11
10	207	5/6/77	10	290	2/11/77	8	170	7/2/77	6

In the model, NO_2 data for 1973 from 26 monitoring stations were combined with population data from Regional Statistical Areas (RSAs) developed by the Southern California Association of Governments and employment data, computed for each RSA from 1970 Census data. A number of receptor points were assigned to each RSA according to the size of the population and the land area. The number of people associated with each receptor point was then computed. For the non-working population, exposure estimates were made by assuming this sub-population to be stationary. In the case of workers, the population was stratified by the residence location and by the working location. Air quality data in the latter case was classified into two time categories: (1) non-working time and (2) working time (weekdays 7 am to 6 pm). Additionally, weekday-weekend differences in exposure were assessed from data on weekday-weekend differences in air quality.

Using the static population assumption, the distribution of the population exposed at various frequencies of standard violations (population-at-risk distribution) were determined for both NO_2 hourly average concentrations and NO_2 daily maximum hourly concentrations. Figure 8-21 shows the distributions of the population exposed at various percentages of days exceeded during three time periods; all the time, weekdays and weekends. It can be seen that the entire population was exposed for a smaller percentage of days during weekends than weekdays. An average person in the Los Angeles AQCR was exposed to NO_2 air pollution above the CAAQS 4.4 percent of the days during weekdays, and only 2.1 percent of the days during the weekends (Table 8-20).

The distribution of the population exposed at various percentages of hours exceeded is shown in Figure 8-22. Again, the entire population was exposed for a smaller percentage of hours above the CAAQS during weekends than weekdays. Therefore, it can be concluded that people in the Los Angeles AQCR were less frequently exposed to a concentration above the CAAQS during weekends than weekdays because of the markedly better NO_2 air quality over weekends.

The regionwide impacts of weekday-weekend phenomena on population exposure to NO_2 are summarized in Table 8-20. The regional averages of daily risk frequency and hourly risk frequency were, respectively, 3.7 percent of the days and 0.46 percent of the hours. In other words, an average person in the Los Angeles Basin was exposed in 1973 to a concentration above the CAAQS 14 days per year or 40 hours per year. The regional averages of daily risk frequency are 4.4 percent of the days during weekdays and 2.1 percent of the days during weekends. The regional averages of hourly risk frequency are 0.57 percent of the hours during weekdays and 0.18 percent of the hours during weekends. Therefore, it can be said that in 1973 an average person in the Los Angeles Basin received a less frequent exposure above the CAAQS during weekends than weekdays by 2.3 percent of the days or by 0.39 percent of the hours.

Table 8-20 also presents the regional averages of risk frequency, which were computed by considering diurnal population movement between residence and workplace (values in parentheses). The refined estimates of regional average risk frequency are close to but a

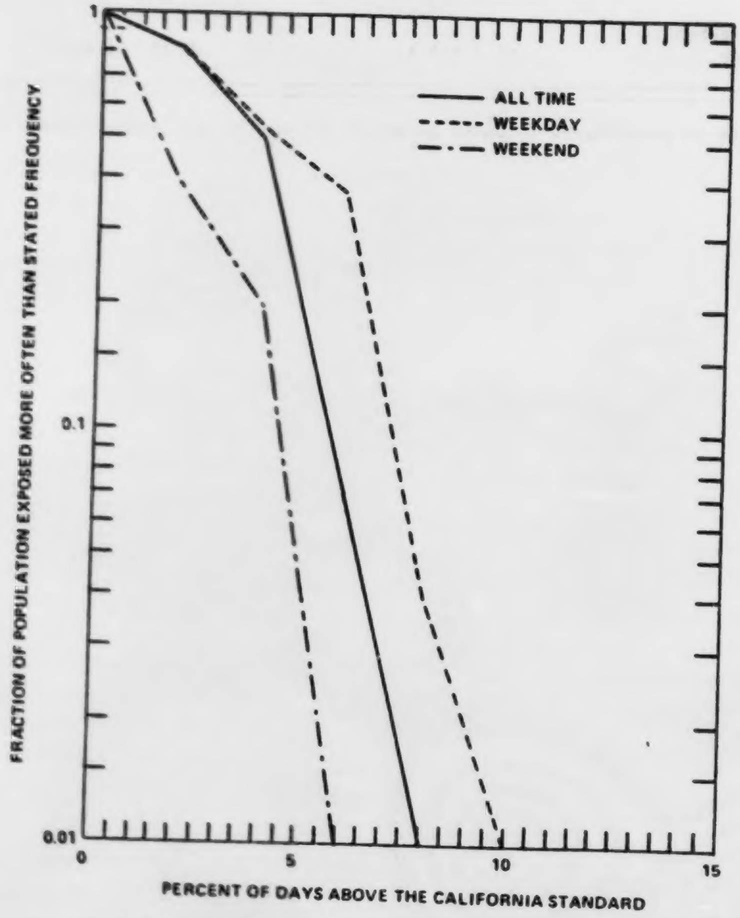


Figure 8-21. Population exposed to NO_2 daily maximum hourly concentration above the California one-hour standard at various frequencies (Horie et al., 1977).

TABLE 8-20. REGIONWIDE IMPACT OF WEEKDAY-WEEKEND PHENOMENA ON POPULATION EXPOSURE TO NITROGEN DIOXIDE: DAYS AND HOURS EXCEEDING THE CALIFORNIA AMBIENT AIR QUALITY STANDARD (FAIRFAX COUNTY)

Time Period	Percent of Days Exceeded[a]	Percent of Hours Exceeded[a]
All time	3.7 (3.8)	0.46 (0.50)
Weekday	4.4 (4.5)	0.57 (0.63)
Weekend	2.1 (2.1)	0.18 (0.18)
Weekday/Weekend Difference	+2.3 (+2.4)	+0.39 (+0.45)

[a] Percentages in parentheses computed based on the mobile population assumption.

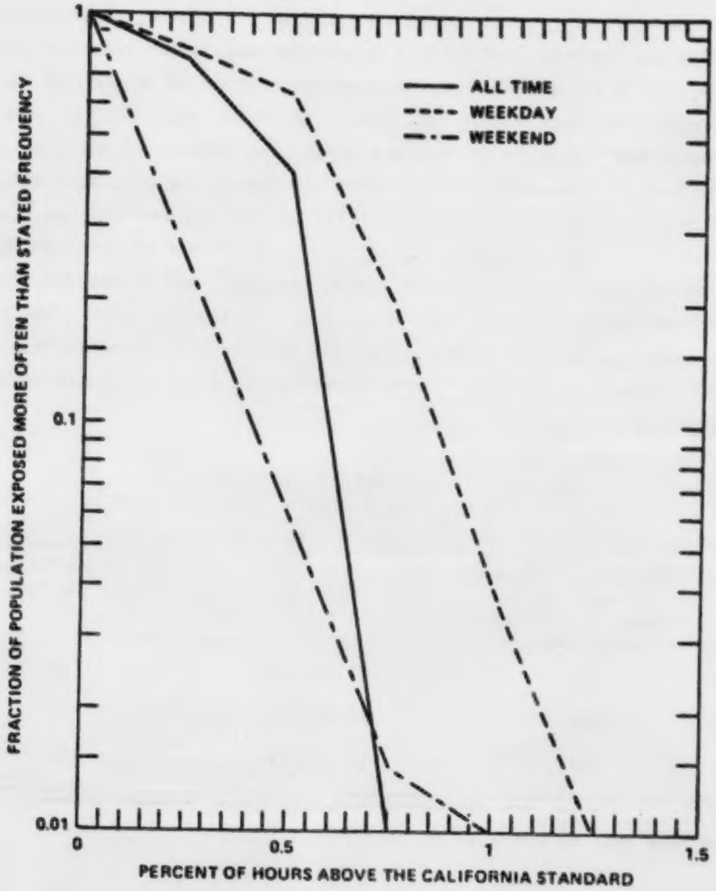


Figure 8-22. Population exposed to NO_2 hourly average concentration above the California one-hour standard (470 $\mu g/m^3$; 0.25 ppm) at various frequencies.

Source: Horie et al., 1977.

little greater than those based on the static population assumption. According to the refined estimates, an average person in the Los Angeles Basin received less frequent exposure above the CAAQS during weekends than weekdays by 2.4 percent of the days or by 0.45 percent of the hours.

A rough nationwide estimate of the population at risk to various levels of NO_2 air pollution can be obtained from a data base recently described (Freedman et al., 1978). The data base includes data on monitoring for all counties in the contiguous United States (taken directly from 1974 SAROAD file) along with 1970 Census data. For the purposes of this analysis, the assumption is made that the entire county population is potentially at risk to the second highest one-hour NO_2 concentration reported in the county, provided that the monitoring site reporting was located specifically to monitor population exposure (SAROAD purpose code 01). In 1974, 121 U.S. counties having total population of 65,009,289 persons had at least one monitor reporting such hourly NO_2 data. Of these counties 112 were selected as having data which warranted inclusion in this analysis. The results of the population-at-risk computation is presented in Table 8-21. Fifty-seven percent of the U.S. population in counties reporting one-hour NO_2 data in 1974 were potentially at risk to one-hour NO_2 concentrations which exceeded 250 $\mu g/m^3$ at least twice during the year; 29 percent to one-hour NO_2 concentrations which exceeded 500 $\mu g/m^3$ at least twice during the year; and 14 percent to one-hour NO_2 concentrations which exceeded 750 $\mu g/m^3$ at least twice during the year. The population at risk comprised at least 41, 21, and 10 million people for the corresponding NO_2 concentration levels. More sophisticated and/or more recent exposure estimates on a nationwide level are lacking in the literature.

TABLE 8-21. U.S. POPULATION AT RISK TO VARIOUS 1974 NO_2 HOURLY AMBIENT CONCENTRATIONS[a]

County Count	1974 Second Highest 1-hour NO_2 Concentration ($\mu g/m^3$)	Total 1970 Population Potentially at Risk	% Monitored Population Potentially at Risk
68	250	41,837,864	57
24	500	21,341,617	29
6	750	10,106,698	14

[a] Computed from data in Freedman et al. (1978).

8.2 ATMOSPHERIC CONCENTRATIONS OF NITRATES

Although extensive monitoring has been carried out for nitrates suspended in ambient air, recent reports (Section 7.4.1) document serious and apparently unresolvable difficulties associated with nitrate artifact formation on the filter media routinely used to collect samples. At present, therefore, it seems most prudent to report data only for those few recent measurements which were collected using Teflon filters not believed to be subject to the positive artifact formation reported.

It should be noted, however, that Harker et al. (1977) report that nitrate could be removed from glass fiber filters when aerosols containing sulfate passed through the filters. Since the same mechanism may operate when Teflon filters are used, the data reported below must be considered as preliminary.

A 24-hr sample from Philadelphia, Pennsylvania, on February 24, 1977, indicated an ambient nitrate concentration of 0.18 $\mu g/m^3$ in the fine particle fraction ($<2.4\mu$) of a sample collected by a dichotomous sampler using Teflon filters (Dzubay and Stevens, 1975; Stevens et al., 1978b). Data from Glendora, California, obtained with a dichotomous sampler during 10 days in early March, 1978, show nitrate concentration ranging from 0.17 to 0.47 $\mu g/m^3$ in the fine fraction with a mean of 0.28 $\mu g/m^3$ (Stevens et al., 1978a). Concentrations from the coarse fraction (particles with aerodynamic diameter between 2.4 μ and 16 μ) range from 0.06 to 0.83 $\mu g/m^3$ with a mean of 0.22 $\mu g/m^3$. Measurements at a number of sites near freeways in Los Angeles gave fine fraction nitrate concentrations up to 2.0 $\mu g/m^3$ and similar readings up to 2.1 $\mu g/m^3$ at nearby background sites (Dzubay et al., 1979). Nitrate did not increase significantly in the roadway (Trijonis, 1978). Other data from California obtained using quartz filters are summarized in Table 7-7 and in Spicer et al. (1978).

Few measurements of ambient nitric acid vapor concentrations have been carried out. Sampling from aircraft in non-urban areas at altitudes ranging from 0.2 to 8 km was conducted by Huebert and Lazrus (1978) during August and September of 1977. Those areas not influenced by urban plumes evidenced concentrations ranging from 0.05 to 0.75 $\mu g/m^3$ (0.02 to 0.3 ppb), with most values below 0.4 $\mu g/m^3$ (0.15 ppb).

Miller and Spicer (1974), using a modified microcoulometric method, report up to 25 $\mu g/m^3$ (10 ppb) HNO_3 in Los Angeles, California. In a more extensive report, Spicer (1977) cites measurements in St. Louis, Missouri, for 26 days during July and August 1973, yielding maximum 23-hr average HNO_3 concentrations of 30 $\mu g/m^3$ (12 ppb) and a 1-hr maximum of 200 $\mu g/m^3$ (80 ppb). In West Corvina, California, 29 sampling days during August and September 1973, gave 23-hr average values up to 65 $\mu g/m^3$ (26 ppb) and a 1-hr maximum of 78 $\mu g/m^3$ (31 ppb). Hanst et al. (1975), in failing to detect HNO_3 in Pasadena, California, set an upper limit of 25 $\mu g/m^3$ (10 ppb) on its concentration in this area. Recently, Tuazon et al. (1978) report observing up to 15 $\mu g/m^3$ (6 ppb) in Riverside, California, during approximately one day of monitoring in October 1976.

The data available are not sufficient to place human exposure to suspended nitrates or nitric acid vapor in nationwide perspective. Furthermore, extreme caution must be exercised in interpreting any health studies making use of ambient nitrate data derived using filter media that are subject to artifact formation.

8.3 ATMOSPHERIC CONCENTRATIONS OF N-NITROSO COMPOUNDS

Although ambient atmospheric data on N-nitroso compounds are sparse when viewed from a nationwide perspective, a number of measurements from scattered locations, mostly near suspected sources, have been reported. Some of these data are presented in this section in order to indicate the possible magnitude of the existing atmospheric burden of this class of compounds.

Fine (1975a,b) first reported dimethylnitrosamine (DMN) in ambient air in 1975. Levels ranged up to 0.95 $\mu g/m^3$ in Baltimore, Maryland, and up to 0.051 $\mu g/m^3$ in Belle, West Virginia. Later, Fine (1976) reported concentrations up to 15 $\mu g/m^3$ near the same site in Baltimore and, independently, Pellizzari (1977) reported values up to 32 $\mu g/m^3$ from the same area using a different analytical method. Fine (1976) reported a level of 0.8 $\mu g/m^3$ for a 3-minute sample taken in New York City, New York. Fine (1975a,b) failed to detect DMN in Philadelphia, Pennsylvania; Waltham, Massachusetts; and at a site near Wilmington, Delaware. Sampling by EPA's National Enforcement Investigations Center (NEIC) (1977b) showed little indication of N-nitroso compounds in the vicinity of suspected sources at 32 locations in Kansas, Missouri, Illinois, Indiana, Iowa, and Nebraska when contamination problems were resolved. No evidence was found by NEIC to substantiate secondary production in the vicinity of amine sources. In other monitoring by NEIC (1977c) near suspected sources in McIntosh, Alabama; Charlotte and Salisbury, North Carolina; and Chattanooga, Tennessee, only one sample near the Alabama site showed evidence of N-nitroso compounds (0.040 $\mu g/m^3$ DMN) in the atmosphere. Fine (1976) reported monitoring for atmospheric DMN at several sites in New York, New Jersey, and Massachusetts under a variety of meteorological conditions. DMN was not found in any of 25 samples taken throughout northern New Jersey; nor in any of 15 samples in the Boston, Massachusetts, area. Only one of 18 samples in New York City showed a measurable DMN concentration (0.016 $\mu g/m^3$). Since a cryogenic trap was used in the sampling procedure for this measurement, the possibility of artifact formation cannot be ruled out.

In summary, these measurements point to the conclusion that the atmospheric route for N-nitroso compounds is not a significant pathway for possible human exposure. In addition, no evidence has been found to date for the nitrosation of amines in ambient air.

8.4 SUMMARY

8.4.1 Atmospheric Concentrations of NO_2

Examination of both historical data and for the years 1975 to 1980 allows some general conclusions to be drawn about the nationwide experience with respect to ambient NO_2 concentrations. In summary, the data cited illustrate the following points.

- Annual average concentrations of NO_2 are not reliable index of short-term (3 hour or less) human exposure, and vice versa.
- Although a distinct recurrent diurnal pattern is discernible in some areas of the country, in many areas peak diurnal values may occur at almost any time of day.
- Just as there is no standard diurnal pattern nationwide for peak NO_2 concentrations, there is also no standard nationwide pattern for the month-by-month variations in monthly averages of daily maximum 1-hr concentrations. Peak monthly averages occur at different times of year in different locations.
- The direction and magnitude of recent trends in NO_2 concentrations also tend to be area-specific.
- The oxidation of NO to NO_2 by ozone scavenging (chemical reaction with ambient O_3, which is generated photochemically during daylight hours) may at some times be an important mechanism in some areas of the country for producing high NO_2 levels after photochemical activity has ceased. In other areas photochemical reactions involving ambient hydrocarbons may be the dominant mechanism.

The following summary of recent ambient levels of NO_2 occurring nationwide is given to provide perspective on the concentration levels associated with the health and welfare effects discussed elsewhere in this document.

Examination of selected nationwide monitoring data for 1975 to 1980 reveals that during at least one of these years, peak 1-hr NO_2 concentrations equalling or exceeding 750 $\mu g/m^3$ (0.4 ppm) were experienced in Los Angeles and several other California sites; Ashland, Kentucky; and Port Huron, Michigan. Additional sites reporting at least one peak hourly concentration equalling or exceeding 500 $\mu g/m^3$ (0.27 ppm) include: Phoenix, Arizona; St. Louis, Missouri; New York City, New York; 14 additional California sites; Springfield, Illinois; Cincinnati, Ohio; and Saginaw and Southfield, Michigan. Other sites, distributed nationwide, reported maxima close to this value. Recurrent NO_2 hourly concentrations in excess of 250 $\mu g/m^3$ (0.14 ppm) were quite common nationwide in 1975 to 1980.

The data also show that Long Beach, California; Indianapolis, Indiana; and Salt Lake City, Utah, all experienced at least one day when the 24-hr average NO_2 concentration exceeded 300 $\mu g/m^3$ (0.16 ppm) during 1975 to 1980. In addition, San Bernadino, California; Denver, Colorado; Chicago, Illinois; Nashua, New Hampshire; Cincinnati and Cleveland, Ohio; Tulsa, Oklahoma; and Fort Worth and Houston, Texas, all reported at least one 24-hr period where average NO_2 concentrations exceeded 150 $\mu g/m^3$ (0.08 ppm).

Annual arithmetic means for NO_2 concentrations in 1976 exceeded 100 $\mu g/m^3$ (0.053 ppm) at Anaheim, El Cajon, Riverside, San Diego, and Temple City, California. Other sites reporting yearly arithmetic means for 1976 equalling or exceeding 100 $\mu g/m^3$ (0.053 ppm) included Chicago, Illinois, and Southfield, Michigan. However, by 1980 virtually none of the still operating monitoring sites reported annual average levels over 100 $\mu g/m^3$ (except one in San Diego; 114 $\mu g/m^3$).

8.4.2 Atmospheric Concentrations of Nitrates

Few high quality data exist on concentrations of nitrates suspended in ambient air. Very recent data, taken by sampling techniques not subject to positive artifact formation, range from 0.18 $\mu g/m^3$ in Philadelphia, Pennsylvania, to 2.1 $\mu g/m^3$ at sites in Los Angeles. The data available are not sufficient to place human exposure in nationwide perspective.

8.4.3 Atmospheric Concentrations of N-Nitroso Compounds

N-nitroso compounds have recently been observed in ambient air, mostly at locations near known or suspected sources. The data reported to date are indicators only of atmospheric burdens at a few special locations. No evidence has been reported to substantiate secondary production in the vicinity of amine sources.

Observed concentrations of dimethylnitrosamine (DMN) ranged up to 32 $\mu g/m^3$ at a site in Baltimore, Maryland, near a known emission source. An extensive monitoring survey by EPA's National Enforcement Investigation Center showed no indication of N-nitrosamines in the vicinity of 32 suspected sources throughout the Midwest. Similar monitoring at four sites in the Southeast yielded a trace of DMN in only one sample from one site. Similar results were obtained in sampling by other researchers in the greater New York-New Jersey area and near Boston, Massachusetts. Considering the small and infrequently observed atmospheric burdens of nitrosamines reported, in conjunction with the potential human exposure from certain foodstuffs and tobacco, the atmospheric route for human exposure does not, at this time, seem to be a significant one.

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9. PERTURBATIONS OF THE STRATOSPHERIC OZONE LAYER

9.1 INTRODUCTION

Since the beginning of this decade it has been increasingly clear that a number of man's activities can lead to reductions in stratospheric ozone, which protects life at the earth's surface from potentially harmful ultraviolet radiation. Initially attention was directed to the pollution of the stratosphere by direct injections of water vapor and oxides of nitrogen (NO and NO_2) from high-flying aircraft (Crutzen, 1971; Johnston, 1971). It had been proposed by Crutzen (1970) that NO_x ($NO + NO_2$) could catalyze the destruction of ozone and control its stratospheric abundance by the set of photochemical reactions:

$$O_3 + h\nu \rightarrow O + O_2 \qquad (9\text{-}1)$$

(wavelengths shorter than 1140 nm)

$$O + NO_2 \rightarrow NO + O_2 \qquad (9\text{-}2)$$

$$\underline{NO + O_3 \rightarrow NO_2 + O_2} \qquad \underline{(9\text{-}3)}$$

$$2\,O_3 \rightarrow 3\,O_2 \qquad \text{net}$$

The main source of NO_x in the stratosphere is probably the oxidation of nitrous oxide (N_2O) via the reactions (Crutzen, 1971; Nicolet and Vergison, 1971; McElroy and McConnell, 1971):

$$O_3 + h\nu \rightarrow O(^1D) + O_2 \qquad (9\text{-}4)$$

(wavelengths shorter than 310 nm)

$$O(^1D) + N_2O \rightarrow 2\,NO \qquad (9\text{-}5)$$

Because of its relatively low solubility and low reactivity in water, nitrous oxide is not removed in clouds and through precipitation in the troposphere. In contrast, direct transport of NO and NO_2 into the stratosphere from the earth's surface is strongly prohibited by wet removal of NO_2 and especially its oxidation product HNO_3, which is formed by the reaction:

$$OH + NO_2\ (+M) \rightarrow HNO_3\ (+M) \qquad (9\text{-}6)$$

The hydroxyl radical (OH) is primarily formed by the attack of $O(^1D)$ on water vapor, following reaction 9-4, above:

$$O(^1D) + H_2O \rightarrow 2\,OH \qquad (9\text{-}7)$$

This radical, and therefore also ozone, plays an extremely important role in the photochemistry of the atmosphere. In the troposphere (0-10 km in middle and high latitudes, or 0-18 km in the tropics) it attacks a host of gases which otherwise would be inert, such as CO, H_2, hydrocarbons and chlorinated hydrocarbons. This ensures that only a small portion (~10% or less) of the ground level emissions of such gases can reach the stratosphere. As a result, photochemical activity in the stratosphere is strongly limited and ozone is protected from otherwise larger destruction. Nitrous oxide, however, is nearly inert to attack by all known tropospheric gases, including OH (Biermann et al., 1976).

9.2 DIRECT ROLE OF NITROGEN OXIDES IN THE OZONE BALANCE OF THE ATMOSPHERE

The oxides of nitrogen, NO_x, play a remarkable catalytic role in the ozone balance of the atmosphere. Above about 24 km, the net effect of NO_x additions to the stratosphere is ozone destruction by the set of reactions already discussed (reactions 9-1, 9-2, 9-3). At lower altitudes the opposite is true. The essential reason for this is the following set of reactions.

$$\begin{array}{ll}
RO_2 + NO \rightarrow RO + NO_2 & (9\text{-}8) \\
NO_2 + h\nu \rightarrow NO + O & (9\text{-}9) \\
\underline{O + O_2 + M \rightarrow O_3 + M} & \underline{(9\text{-}10)} \\
RO_2 + O_2 \rightarrow RO + O_3 & \text{net}
\end{array}$$

This is the same set of reactions which is at the core of ozone production under photochemical smog conditions, when reactions 9-8 through 9-10 are preceded by reactions of the type

$$R + O_2 + M \rightarrow RO_2 + M \qquad (9\text{-}11)$$

with the net result: $R + 2\,O_2 \rightarrow RO + O_3$. The radical R can here stand for such radicals as CH_3CO and H. In the non-urban troposphere, ozone production may, however, still take place from the oxidation of hydrocarbons emitted by vegetation and of carbon monoxide, provided sufficient NO_x is present. In the case of carbon monoxide the sequence of reactions starts with

$$CO + OH\ (+M) \rightarrow H + CO_2\ (+M) \qquad (9\text{-}12)$$

followed by reaction 9-11 (with R = H) and reactions 9-8 through 9-10, leading to the net result: $CO + 2\,O_2 \rightarrow CO_2 + O_3$. Because it is conceivable that both carbon monoxide and nitric oxide concentrations have been increasing due to human activities, there is a discrete possibility of worldwide ozone increases, especially in the Northern Hemisphere's troposphere (Fishman and Crutzen, 1978).

It should be added that the origin of tropospheric ozone is currently not well known. Traditional thinking in the meteorological community explained the presence of ozone in the troposphere by its formation in the stratosphere (Chapman, 1930) via the reaction

$$\begin{array}{ll}
O_2 + h\nu \rightarrow 2\,O \quad (\lambda < 240\ \text{nm}) & (9\text{-}13) \\
\underline{O + O_2 + M \rightarrow O_3 + M \quad (\times 2)} & \underline{(9\text{-}14)} \\
3\,O_2 \rightarrow 2\,O_3 & \text{net}
\end{array}$$

and its downward transport into the troposphere in the vicinity of frontal zones or tropopause breaks (Danielsen and Mohnen, 1977). The tropospheric ozone production taking place near urban centers was thought to be only of minor global importance. However, it seems now that ozone production can also take place in the "clean" troposphere as long as NO_x volume mixing ratios are not too small ($\geq 10^{-11}$), because of the fast rate of reaction 9-8.

In the lower stratosphere (~10-24 km) the chain of reactions 9-8 through 9-10 (with R = H) tends to counteract the effect of the reactions

$$OH + O_3 \rightarrow HO_2 + O_2 \qquad (9\text{-}15)$$

$$HO_2 + O_3 \rightarrow OH + 2\,O_2 \qquad (9\text{-}16)$$

$$2\,O_3 \rightarrow 3\,O_2 \qquad \text{net}$$

by deferring it into the sequence

$$OH + O_3 \rightarrow HO_2 + O_2 \qquad (9\text{-}15)$$

$$HO_2 + NO \rightarrow OH + NO_2 \qquad (9\text{-}8)$$

$$NO_2 + h\nu \rightarrow NO + O \qquad (9\text{-}9)$$

$$O + O_2 + M \rightarrow O_3 + M \qquad (9\text{-}10)$$

$$\text{no net chemical effect} \qquad \text{net}$$

Additions of NO to the lower stratosphere, therefore, tend to increase local ozone concentrations by causing smaller ozone losses. The importance of the ozone-producing aspects of NO_x catalysis below about 24 km is dramatically emphasized by the recent discovery of Howard and Evenson (1977), who found reaction 9-8 to be at least an order of magnitude faster than previously determined through indirect measurements of the rate constant. This finding has resulted in substantial downward revisions of estimated total ozone column reductions due to stratospheric NO_x additions from high-flying aircraft. As a result of the peculiar photochemical action of NO_x, we notice also a decrease in altitude of the center of gravity of stratospheric ozone by transfer of mass from above 24 km to below 24 km. As NO_x is produced by the oxidation of N_2O via reactions 9-4 and 9-5, the same conclusions are valid regarding the possible effects of a future rise in the atmospheric content of nitrous oxide. Such an increase may be caused by man's intervention in the nitrogen cycle through an increasing use of nitrogen fertilizer and other agricultural activities.

9.3 INDIRECT ROLE OF NITROUS OXIDE IN THE OZONE BALANCE OF THE ATMOSPHERE

In addition to the direct effects to the stratospheric ozone layer caused by the catalytic cycle involving the nitrogen oxides, the existence of NO_x also has an important influence on the impact of catalytic ozone destruction initiated by other atmospheric constituents. In particular, catalytic destruction of stratospheric ozone via reactive chlorine species proceeds as follows:

$$O_3 + h\nu \rightarrow O + O_2 \qquad (9\text{-}17)$$

$$O + ClO \rightarrow Cl + O_2 \qquad (9\text{-}18)$$

$$O_3 + Cl \rightarrow ClO + O_2 \qquad (9\text{-}19)$$

$$2\,O_3 \rightarrow 3\,O_2 \qquad \text{net}$$

The initial release of Cl (and subsequent formation of ClO) to the stratosphere occurs when degradation of chlorinated compounds such as chlorofluoromethanes (e.g., fluorocarbon-11 and fluorocarbon-12), methyl chloroform, and methyl chloride takes place by either direct photolysis or attack by reactive species which prevail in the stratosphere (Molina and Rowland, 1974). However, the ozone-destroying frequencies of these catalytic cycles are not additive since the presence of both reactive chlorine and reactive nitrogen compounds may result in a short-circuit of either catalytic cycle. For example, the formation of chlorine nitrate in the stratosphere through the reaction

$$ClO + NO_2 + M \rightarrow ClONO_2 + M \qquad (9\text{-}20)$$

removes both reactive chlorine and nitrogen from the atmosphere which would have otherwise been able to take part in catalytic ozone-destroying cycles. As a result, the inclusion of $ClONO_2$ in stratospheric models of ozone depletion predictions lowered the depletion estimates by about a factor of two (Crutzen et al., 1978).

Lastly, it should be pointed out that new measurements of reaction rates which involve only reactive nitrogen species can likewise influence other catalytic cycles of ozone destruction. A good example of such an occurrence is the recently reported rate constant for the reaction

$$HO_2 + NO \rightarrow OH + NO_2 \qquad (9\text{-}8)$$

by Howard and Evenson (1977), which was shown to proceed much faster than had been previously believed (Hampson and Garvin, 1975). An increase in this rate constant increases the $\frac{OH}{HO_2}$ ratio in the model calculations. Because more OH is calculated by the numerical models, smaller concentrations of HCl are computed to exist. Since HCl is one of the primary reservoirs of reactive chlorine species, the increased OH concentrations which have been brought about by the reaction of NO with HO_2, result in the release of more chlorine atoms into the atmospheric system via

$$HCl + OH \rightarrow H_2O + Cl \qquad (9\text{-}21)$$

Thus, it is important to point out that the nitrogen cycle in the stratosphere has a direct bearing on the catalyzing power of other stratospheric cycles involving the ozone layer. This interaction may be interferring or enhancing depending on which region of the ozone layer is being examined or at which altitudes certain reactions dominate the overall chemistry.

9.4 OTHER ATMOSPHERIC EFFECTS OF NITROGENOUS COMPOUNDS

The environmental effects of ozone changes in the stratosphere are, however, not solely related to the function of ozone as a shield against the penetration of solar ultraviolet radiation to ground level. Ozone is also an important gas for the heat budget of the atmosphere. An effective lowering of the center of gravity of the ozone layer, which would be the result of NO_x additions to the stratosphere, may have significant climatic implications (Ramanathan, 1974), as it may cause a warming of the lower stratosphere and the earth's surface by the increased absorption of ultraviolet solar radiation and enhanced trapping of thermal 9.6 μm radiation emitted from the warm surface of the earth.

The significance of nitrous oxide is also not restricted to its importance as a source of stratospheric NO_x via reactions 9-4 and 9-5. Because of its absorption bands at about 7.8 μm and 17.0 μm, nitrous oxide (N_2O) likewise contributes significantly to the atmospheric "greenhouse" effect by trapping outgoing terrestrial radiation. It has, therefore, been estimated that a doubling of the atmospheric N_2O content could cause an increase in surface temperatures by as much as 0.7^oK (Wang et al., 1976). Several recent studies have been designed to estimate the possible extent of future atmospheric N_2O build-up due to increased use of nitrogen fertilizer (CAST, 1976; Crutzen, 1976; Crutzen et al., 1976; Liu et al., 1976; McElroy, 1974; McElroy et al., 1977; Rice and Sze, 1976; Sze and Rice, 1976). It is difficult, however, to estimate the doubling time with any certainty. Three important but poorly understood factors determining the release of N_2O from soil and water to the atmosphere are the following:

1. The release of N_2O in the denitrification process. This microbiological process, which is currently considered to be the main source of atmospheric N_2O, takes place in anaerobic microenvironments, and involves the reduction of nitrate to N_2O and molecular nitrogen (N_2). It is this process which presumably balances nitrogen fixation, i.e., the conversion of N_2 to fixed nitrogen. A growing amount of observational evidence is now accumulating, which indicates that the yield of N_2O versus N_2 in agricultural and water environments is less than 20 percent (Delwiche, 1977; Rolston, 1977). It remains, however, important to gather additional information from field studies to improve the data base for this important environmental factor.
2. The pathway of fertilizer nitrogen in the environment. This involves knowledge of such factors as the actual amount of denitrification in agricultural fields, leaching of nitrate to groundwater, volatilization of ammonia and its transport and conversions in the atmosphere, the cycling and decomposition of animal manures in the environment, and the extent of transfer of agricultural fixed nitrogen to the natural ecosystems, which may be expected to have much longer turnover times than those systems which are directly affected by agriculture. Studies of these matters have been conducted by Liu et al. (1976), McElroy et al. (1977), and Crutzen and Ehhalt (1976).
3. The role of the oceans in the worldwide N_2O budget. While initial studies indicated a large source of N_2O in the oceans (National Academy of Sciences, 1978), recent investigations point towards a much smaller role of oceans in the global N_2O budget (Hahn, 1974; Weiss, 1977).

The scientific problems connected with possible future increases in atmospheric N_2O concentrations are being investigated by several research groups and the issue has been reviewed in a recently published report by an ad hoc committee of the National Academy of Sciences (1978). The issue is further complicated by the fact that the nitrogen cycle is coupled to other cycles, such as those of carbon and phosphorus (Simpson et al., 1977). It is, therefore, fair to say that many years of active research are needed to assess reliably this potentially important global environmental issue.

9.5 SUMMARY

Stratospheric ozone protects life at the earth's surface from potentially harmful ultraviolet radiation from the sun. The main source of NO_x in the stratosphere is the oxidation of nitrous oxide (N_2O). Nitrous oxide is ubiquitous, even in the absence of human activities, since it is a product of natural biologic processes in soil, but significant quantities may also arise from the denitrification of the increased quantities of fixed nitrogen, which are introduced into the nitrogen cycle by the growing use of nitrogen fertilizers. Recent reports indicate that somewhat less than 20 percent of the "excess" nitrogen eventually escapes as N_2O, with most of the rest returned to the atmosphere as N_2. Since N_2O is not believed to take part in any atmospheric chemical reactions below the stratosphere, all the N_2O produced terrestrially is available for stratospheric reactions. The concern expressed by some authors in the recent past, that N_2O arising from excess fertilizer might decrease the total stratospheric ozone by as much as 20 percent for a 100 percent increase in total N_2O, has recently been reevaluated in the light of new information on rate constants for certain stratospheric chemical reaction pathways. These new considerations point to the likelihood of a much smaller dependence of total stratospheric ozone on N_2O abundance.

Ozone is also an important gas for the heat budget of the atmosphere. An effective lowering of the "center of gravity" of the ozone layer, which would be the result of NO_x additions to the stratosphere, may have significant climatic implications. Nitrous oxide likewise contributes to the atmospheric "greenhouse" effect by trapping outgoing terrestrial radiation. One author recently estimated that a doubling of the atmospheric burden of N_2O could increase surface temperatures by as much as 0.7°C. It is difficult, however, to estimate the doubling time with any certainty. Since global estimates of the end effects of excess fertilizer rest on a number of poorly known parameters, and since the issues are further complicated by the fact that the nitrogen cycle is coupled to other cycles, such as the carbon cycle, no definitive conclusions can prudently be drawn.

CHAPTER 9

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10. EFFECTS OF NITROGEN OXIDES ON VISIBILITY

Air pollution degrades the appearance of distant objects and reduces the range at which they can be distinguished from the background. These effects are manifested not only in visible plumes, but also in large-scale, hazy air masses (Husar et al., 1976). Haze and plumes can result in the deterioration and loss of scenic vistas, particularly in areas such as the southwestern U.S. where visibility is generally good. Under extreme conditions, reduced visual range and contrast due to haze and plumes may impede air traffic. NO_2 can be responsible for a portion of the brownish coloration often observed in polluted air (Hodkinson, 1966; Husar and White, 1976). However, it should also be noted that non-nitrate particulate matter has been implicated in the production of a significant fraction of brownish coloration (Husar and White, 1976). In addition, where nitrates occur as fine particles, they may contribute to the reduction of visual range (White and Roberts, 1977; Trijonis and Yuan, 1978b). (See Chapter 6 for a discussion of atmospheric processes resulting in ambient burdens of NO_2 and particulate nitrate.)

10.1 NITROGEN OXIDES AND LIGHT SCATTERING AND ABSORPTION

Deterioration in visibility is due to the absorption and scattering of light by gaseous molecules and suspended solid or liquid particles (Middleton, 1952). Absorbed light is transformed into other forms of energy, such as heat, while scattered light is re-radiated in all directions with no change in wavelength. The importance of each process is determined by the absorption and scattering coefficients, b_a and b_s. These coefficients specify the rate at which a beam of light is attenuated in passing through the atmosphere:

$$dI_a/I = -b_a dx, \quad \text{and} \quad dI_s/I = -b_s dx,$$

where I is the intensity of the beam, and dI_a and dI_s are the changes in I due to absorption and scattering over the incremental distance dx. The sum of the absorption and scattering coefficients is the total extinction coefficient, $b = b_a + b_s$.

The absorption and scattering coefficients of particulate matter and the different gases are additive. It is, therefore, meaningful to speak of the impact of an individual species on visibility. The effects of gases and particulate matter can be distinguished as follows (the subscripts g and p denote the respective contributions from gases and particulates):

$$\begin{aligned} b &= b_a + b_s \\ &= b_{ag} + b_{ap} + b_{sg} + b_{sp} \end{aligned}$$

In polluted atmospheres, the term b_{ag} is dominated, at visible wavelengths, by the contribution from NO_2, which absorbs strongly in the blue region of the spectrum. The scattering due to trace gases is negligible, so that b_{sg} can be regarded as the constant background due to Rayleigh scattering by clean air. Absorption and scattering by particles depend on their size and composition (U.S. DHEW, 1969). Nitrate compounds may constitute a significant fraction in the optically important particle size range, but current information on ambient nitrate concentrations is insufficient to make any conclusive assessment.

10.2 EFFECT OF NITROGEN DIOXIDE ON COLOR

Under typical ambient conditions, light scattering dominates total extinction, which is related to reduction of contrast and visual range. The most significant optical effect of NO_2, however, involves discoloration. The absorption coefficient for NO_2 can be used to calculate the visual impact of NO_2 in the atmosphere (Dixon, 1940).

10.2.1 Nitrogen Dioxide and Plumes

Under certain circumstances, brown plumes may be distinguished tens of kilometers downwind of their sources. Nitrogen dioxide in a plume acts as a blue-minus filter for transmitted light. It tends to impart a brownish color to targets, including the sky, viewed through the plume. The strength of this filter effect is determined by the integral of NO_2 concentration along the sight path; e.g., theoretically similar effects would be produced by a 1 kilometer-wide plume containing 0.1 ppm (190 $\mu g/m^3$) NO_2 or a 0.1 kilometer-wide plume containing 1.0 ppm (1,900 $\mu g/m^3$) NO_2.

Figure 10-1 shows the calculated transmittance of particle-free NO_2 plumes for several values of the concentration-distance product. Less than 0.1 ppm-km NO_2 is sufficient to produce a color shift which is distinguishable in carefully-controlled, color-matching tests (MacAdam, 1942). Reports from one laboratory using NO_2-containing sighting tubes indicate a visible color threshold of 0.06 ppm-km for the typical observer. This value was supported by a few field observations of NO_2 plumes from nitric acid manufacturing plants under varying operating conditions (Hardison, 1970). The value cited refers to the effect of NO_2 in the absence of atmospheric aerosol. Empirical observations under a variety of conditions are needed to determine the perceptibility of NO_2 in ambient air.

Discoloration of plumes and haze layers by NO_2 is modified by particulate matter and also depends on a number of factors such as sun angle, surrounding scenery, sky cover, viewing angle, human perception parameters, and pollutant loading. The relative importance of aerosol or NO_2 in determining the color and appearance of a plume or haze layer can be addressed, in part, in terms of the relative extinction as a function of wavelength. Suspended particles generally scatter much more in the forward direction than in any other direction. This fact means a plume or haze layer can appear bright in forward scatter (sun in front of the observer) and dark in back scatter (sun in back of the observer) because of the angular variation in scattered air light. This effect can vary with background sky and objects. Aerosol optical effects alone are capable of imparting a reddish brown color to a haze layer when viewed in backward scatter. NO_2 would increase the degree of coloration in such a situation (Ahlquist and Charlson, 1969; Charlson et al., 1978). When the sun is in front of the observer, however, light scattered toward him by particles in the plume tends to wash out the brownish light transmitted from beyond. Under these conditions, particle scattering diminishes the plume coloration. Specific circumstances of brown layers must be observed on a case-by-case basis.

10.2.2 Nitrogen Dioxide and Haze

A common feature of pollutant haze is its brown color. The discoloration of the horizon sky due to NO_2 absorption is determined by the relative concentrations of NO_2 and light-

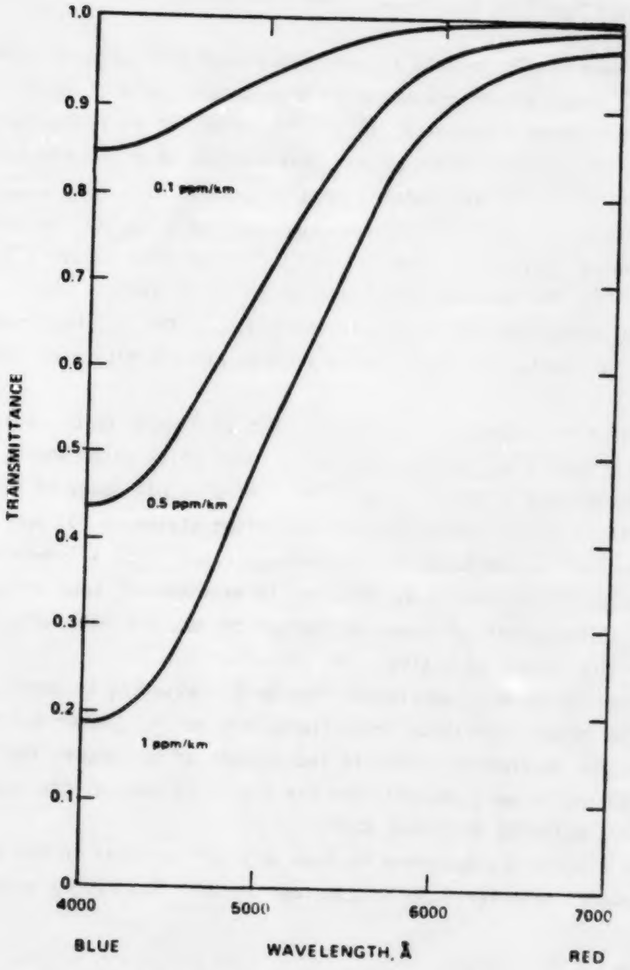

Figure 10-1. Transmittance exp(-$b_{NO_2}x$) of NO_2 plumes for selected values of the concentration-distance product (Adapted from Hodkinson, 1966.)

scattering particles. In a uniform atmosphere, the effect of NO_2 at any given wavelength is described by the following relationship (Robinson, 1968):

$$B_{horizon}/B_{horizon}([NO_2] = 0) = \frac{b_s}{b_s + b_{NO_2}} = (1 + b_{NO_2}/b_s)^{-1}$$

where $B_{horizon}$ and $B_{horizon}([NO_2] = 0)$ are the brightness of the horizon sky, with and without NO_2.

The ratio bNO_2/b_s is more easily related to experience when expressed in terms of concentration, $[NO_2]$, and visual range, VR. As discussed in Section 10.3, visual range under certain simplifying assumptions is inversely proportional to extinction, which is typically dominated by scattering. Samuels et al. (1973) compared human observations with instrumental measurements and found indications that VR and b_s are related by the formula, $VR = (3 \pm 1)/b_s$. Since, in addition, b_{NO2} is proportional to $[NO_2]$, it follows that the ratio b_{NO2}/b_s is proportional to the product $[NO_2]$ VR. Figure 10-2 shows, for several values of this product, the calculated alteration contributed by NO_2 to horizon brightness which is in turn a function of aerosol scattering. It should be noted that this calculation neglects the wavelength dependence of the scattering, which can be substantial in relatively clean air and mitigates the discoloring effects of NO_2.

The interpretation of Figure 10-2 is similar to that of Figure 10-1. A concentration-visual range product of 0.3 ppm-km NO_2 corresponds to a color shift which should be detectable in a polluted layer viewed against relatively clean sky. At a visual range of 100 kilometers, typical of the rural northern great plains area of the United States, 0.003 ppm (6 $\mu g/m^3$) NO_2 might suffice to color the horizon noticeably. At a visual range of 10 kilometers, typical of urban haze, 0.03 ppm (60 $\mu g/m^3$) NO_2 might be required to produce the same effect. However, quantitative theoretical calculations of human perception of NO_2 are not fully developed and experimental observations are needed to evaluate the actual effect.

Independent of absorption by NO_2, wavelength-dependent scattering by small particles can also produce a noticeable brown coloration in polluted air masses (Husar and White, 1976). Unlike the coloration due to absorption, which is independent of sun angle, the brown coloration contributed by scattering is most intense when the sun is in back of the observer.

10.3 EFFECT OF PARTICULATE NITRATES ON VISUAL RANGE

The visual range in a uniform atmosphere is inversely proportional to the extinction coefficient. For a "standard" observer, the Koschmeider formula expressing this relationship is:

$$VR = 3.9/b$$

where b is the extinction coefficient. Taking account of the response as a function of wavelength of the light-adapted eye of a "standard observer" and of the wavelength dependence of

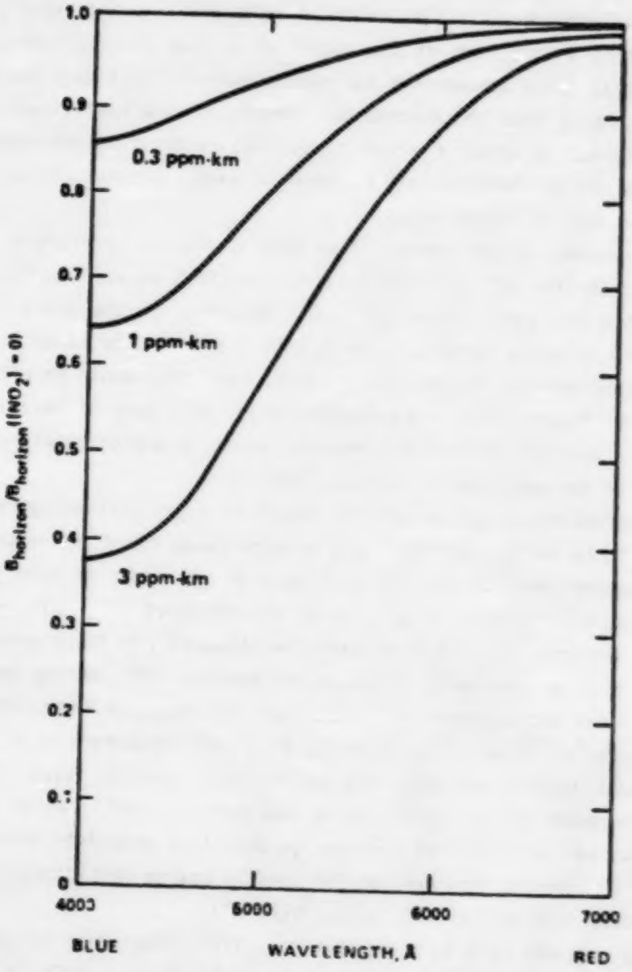

Figure 10-2. Relative horizon brightness, $b_s/(b_s + b_{NO_2})$, for selected values of the concentration-visual product, assuming b_s = 3/(visual range). (Adapted from Hodkinson, 1966.)

Rayleigh scattering, it is possible to compute a mean extinction coefficient in a pure atmosphere containing no aerosols and no light absorbing gases under standard temperature and pressure conditions (0°C, 1.013 bars). The value of b_{sg} so obtained (Penndorf, 1957) is about 0.12 $(10^4 m)^{-1}$, corresponding to a visual range of about 320 km.

Recently, Malm (1979) has examined critically the concept of visual range as a measure of visibility. The simple Koschmeider formula, which generally works reasonably well in urban areas (Horvath and Noll, 1969), is shown to depend upon a number of simplifying assumptions such as: a homogeneous atmosphere, a flat earth, a horizontal viewing aspect, a black target, and a sky radiance which is the same at the object as at the viewing point. Depending upon particular circumstances, these assumptions may have a marked effect upon the relation between the visual range calculated from the Koschmeider formula and the real visual range defined as the distance from a target at which a given (threshold) contrast is achieved. Malm suggests that visibility may be better characterized by apparent target contrast or by color changes of selected vistas rather than by visual range.

A definitive assessment of the contribution made by nitrate aerosols to total extinction (and therefore to degradation of visibility) is not possible because sufficient high-quality data are not available for particulate nitrates. However, to the extent that particulate nitrates are secondary aerosols formed in the 0.1 to 1 micron size range (Lee and Patterson, 1969), it would be expected, on theoretical grounds, that they would be efficient scatterers of light. As shown in Figure 10-3, light scattering per unit mass of aerosol exhibits a pronounced resonance at a particle size of 0.5 microns, which is approximately equal to the wavelength of the center of the spectrum of visible light.

Theoretical calculations based on the Mie theory of light scattering from aerosols indicate that particles found in the 0.1 to 1 micron size range (such as in secondary aerosols) should exhibit extinction coefficients per unit mass on the order of 0.06 ± 0.03, where the units are $(10^4 m)^{-1}/(\mu g/m^3)$ (Latimer et al., 1978; Ursenback et al., 1978; White and Roberts, 1977). Similar calculations indicate that particles occurring in the coarse size range above 2 microns, such as dust or sea spray (Bradway and Record, 1976; Whitby and Sverdrup 1978), should exhibit much lower extinction coefficients per unit mass, on the order of 0.006 ± 0.003 where the units are $(10^4 m)^{-1}/(\mu g/m^3)$ (Latimer et al., 1978; Ursenback et al., 1978; White and Roberts, 1977). These results are confirmed by empirical studies (Cass, 1976; Trijonis and Yuan, 1978a,1978b; Waggoner et al., 1976; White and Roberts, 1977), which typically find extinction coefficients per unit mass of sulfates (a prevalent secondary aerosol) to be 0.04 to 0.10 $(10^4 m)^{-1}/(\mu g/m^3)$. For the remainder of TSP (mostly coarse particles), the extinction coefficient per unit mass is 0.004 to 0.01 $(10^4 m)^{-1}/(\mu g/m^3)$.

Because nitrate aerosols tend to be hygroscopic, light-scattering per unit mass of nitrate can rise significantly with increasing relative humidity (Covert, 1974; Hidy et al., 1974). As relative humidity increases, the mass of water attached to nitrate particles increases and corresponding shifts in the particle size distribution occur. These effects are especially

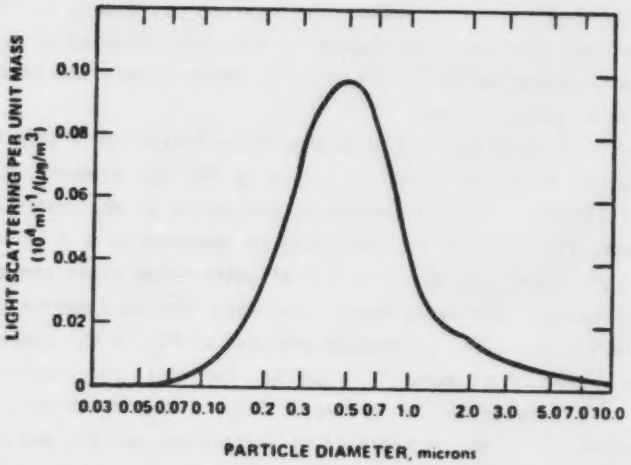

Figure 10-3. Normalized light scattering by aerosols as a function of particle diameter. Computed for unit density spherical particles of refractive index 1.5 (White and Roberts, 1977).

pronounced at relative humidities above 70%; at very high relative humidities (90-100%), rather small shifts in relative humidity may produce pronounced changes in the amounts of light-scattering from a fixed amount of nitrate aerosol.

Even though nitrate aerosols are suspected of occurring in the optically critical 0.1 to 1 micron size range, and even though light-scattering by nitrates may be significantly augmented by relative humidity effects, the actual contributions of nitrate to haze levels may not be significant because ambient nitrate levels may be very low (see data in Chapter 8). A complete characterization of the role nitrates play in visibility degradation must wait until high-quality data bases on nitrates are generated.

10.4 SUMMARY

Air pollution degrades the appearance of distant objects and reduces the range at which they can be distinguished from the background. These effects are manifest not only in visible plumes, but also in large-scale, hazy air masses. Haze and plumes can result in the deterioration and loss of scenic vistas, particularly in areas of the southwestern United States where visibility is generally good. Under extreme conditions reduced visual range and contrast due to haze and plumes may impede air traffic. NO_2 can be responsible for a portion of the brownish coloration observed in polluted air. However, it should be noted that non-nitrate particulate matter has also been implicated in the production of a significant portion of brownish coloration. Under certain circumstances, brown plumes may be distinguished tens of kilometers downwind of their sources.

Nitrogen dioxide in a plume acts as a blue-minus filter for transmitted light. It tends to impart a brownish color to targets, including the sky viewed through the plume. The strength of this filter effect is determined by the amount of NO_2 concentration along the sight path; i.e., theoretically similar effects would be produced by a 1 kilometer-wide plume containing 0.1 ppm (190 $\mu g/m^3$) of NO_2 or a 0.1 kilometer-wide plume containing 1.0 ppm (1,900 $\mu g/m^3$) of NO_2. Based on laboratory tests plus very limited supporting observations in the field, the visible threshold for coloration produced by NO_2 in the atmosphere might be a concentration-distance product of about 0.06 ppm-km. Empirical observations under a variety of conditions are needed to determine the perceptibility of NO_2 in ambient air.

Plume coloration due to NO_2 is modified by particulate matter, and depends on a number of factors such as sun angle, surrounding scenery, sky cover, viewing angle, human perception parameters, and pollutant loading. Suspended particles generally scatter in the forward direction, and can thus cause a haze layer or a plume to appear bright in forward scatter (sun in front of the observer) and dark in back scatter (sun in back of the observer) because of the angular variation in scattered air light. This effect can vary with background sky and objects. Aerosol optical effects alone are capable of imparting a reddish brown color to a haze layer when viewed in backward scatter. NO_2 would increase the degree of coloration in such a situation. When the sun is in front of the observer, however, light scattered toward him by particles in the plume tends to wash out the brownish light transmitted from beyond.

Under these conditions, particle scattering diminishes the plume coloration. Estimates of the magnitude of this effect attributable to particulate nitrates are currently hampered by the lack of data on particulate nitrate concentrations in ambient air.

Nitrogen dioxide and particulate nitrates may also contribute to pollutant haze. The discoloration of the horizon sky due to NO_2 absorption is determined by the relative concentrations of NO_2 and light-scattering particles. A concentration-visual range product of 0.3 ppm-km NO_2 corresponds to a color shift which should be detectable in a polluted layer viewed against a relatively clean sky. At a visual range of 100 km, typical of the rural U.S. great plains, 0.003 ppm (6 μ/m^3) NO_2 might suffice to color the horizon noticeably. At a visual range of 10 km, typical of urban haze, 0.03 ppm (60 $\mu g/m^3$) NO_2 might be required to produce the same effect. For the reason cited above, no reasonable estimate of particulate nitrate contribution to this phenomenon can currently be made. Similarly, an assessment of the role of nitrate aerosols in the degradation of visual range must await the availability of a sufficient data base on ambient particulate nitrate concentrations.

CHAPTER 10

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11. ACIDIC DEPOSITION

11.1 INTRODUCTION

The occurrence of acidic precipitation in many regions of the United Sates, Canada, northern Europe, Taiwan and Japan has become a major environmental concern. Acidic precipitation in the Adirondack Mountains of New York State, in the eastern Precambrian Shield area of Canada, in southern Norway and in southwest Sweden has been associated with the acidification of waters in ponds, lakes and streams with a resultant disappearance of animal and plant life. Acidic precipitation (rain and snow), also is believed to have the potential for leaching elements from sensitive soils, causing direct and indirect injury to forests. It also has the potential for damaging monuments and buildings made of stone, for corroding metals and for deteriorating paint.

The story of acidic precipitation is an ever-changing one. New information concerning the phenomenon is forthcoming nearly every day. The sections that follow emphasize the effects of wet deposition of sulfur and nitrogen oxides and their products on aquatic and terrestrial ecosystems. Dry deposition also plays an important role, but contributions by this process have not been quantified. Because sulfur and nitrogen oxides are so closely linked in the formation of acidic precipitation, no attempt has been made to limit the discussion which follows to the main topic of this document, nitrogen oxides.

Chapter 12 emphasizes the effects of the dry deposition of nitrogen oxides on vegetation and ecosystems. The sources and emissions of nitrogen oxides are discussed in Chapter 5 and those of sulfur oxides in Air Quality Criteria for Particulate Matter and Sulfur Oxides. Chapter 6 discusses the transformation and transport of nitrogen oxides. Ambient air concentrations are discussed in Chapter 8, and the nitrogen cycle in Chapter 4.

11.1.1 Overview of the Problem

The generally held hypothesis is that sulfur and nitrogen compounds are largely responsible for the acidity of precipitation. The emissions of the sulfur and nitrogen compounds involved in acidification are attributed chiefly to the combustion of fossil fuels. Emissions may occur at ground level, as from automobile exhausts, or from stacks of 1000 feet or more in height. Emissions from natural sources are also involved; however, in highly industrialized areas, emissions from man-made sources well exceed those from natural sources. In the eastern United States the highest emissions of sulfur oxides are from electric power generators using coal, while in the West, emissions of nitrogen oxides, chiefly from automotive sources, predominate.

The fate of sulfur and nitrogen oxides, as well as other pollutants emitted into the atmosphere, depends on their dispersion, transport, transformation and deposition. Sulfur and nitrogen oxides may be deposited locally or transported long distances from the emission sources. Therefore, residence time in the atmosphere will be brief if the emissions are deposited locally or may extend to days or even weeks if long range transport occurs. The

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chemical form in which emissions ultimately reach the receptor is determined by the complex chemical transformations that take place between the emission sources and the receptor. Long range transport over distances of hundreds or even thousands of miles allows time for a greater number of chemical transformations to occur.

Sulfates and nitrates are among the products of the chemical transformations of sulfur and nitrogen oxides. Ozone and other photochemical oxidants are believed to be involved in the chemical processes that form them. When sulfates and nitrates combine with atmospheric water, dissociated forms of sulfuric (H_2SO_4) and nitric (HNO_3) acids result. When these acids are brought to earth in rain and snow, acidic precipitation occurs. Because of long range transport, acidic precipitation in a particular state or region can be the result of emissions from sources in states or regions many miles away, rather than from local sources. To date, however, the complex nature of the chemical transformation processes has not made it possible to demonstrate a direct cause and effect relationship between emissions of sulfur and nitrogen oxides and the acidity of precipitation.

Acidic precipitation is arbitrarily defined as precipitation with a pH less than 5.6. This value has been selected because precipitation formed in a geochemically clean environment would have a pH of approximately 5.6 due to the combining of carbon dioxide in the air with water to form carbonic acid. Acidity of solutions is determined by the concentration of hydrogen ions (H^+) present and is expressed in terms of pH units--the negative logarithm of the concentration of hydrogen ions. The pH scale ranges from 0 to 14, with a value of 7 representing a neutral solution. Solutions with values less than 7 are acidic, while values greater than 7 are basic. Because pH is a logarithmic scale, a change of one unit represents a tenfold change in acidity, hence pH 3 is ten times as acidic as pH 4. Currently the acidity of precipitation in the northeastern United States normally ranges from pH 3.0 to 5.0; in other regions of the United States precipitation episodes with a pH as low as 3.0 have been reported. For comparison, the pH of some familiar substances are: cow's milk, 6.6; tomato juice, 4.3; cola (soft drink) 2.8, and lemon juice, 2.3.

The pH of precipitation can vary from event to event, from season to season and from geographical area to geographical area. Substances in the atmosphere can cause the pH to shift by making it more acidic or more basic. Dust and debris swept up in small amounts from the ground into the atmosphere may become components of precipitation. In the West and Midwest soil particles tend to be more basic, but in the eastern United States they tend to be acidic. Gaseous ammonia from decaying organic matter makes precipitation more acidic, so in areas where there are large stockyards or other sources of organic matter, acidic precipitation would be more likely to occur.

In the eastern United States sulfur oxide emissions are greater than nitrogen oxides, therefore, sulfates are greater contributors to the formation of acids in precipitation in this region. The ratio between the two emissions, however, has been decreasing. Sulfate concentrations are greater in summer than in winter in the eastern United States. In California, however, around some of the larger cities, nitrates contribute more to the formation of acidity in rainfall. In coastal areas sea spray strongly influences percipitation chemistry by contributing calcium, potassium, chlorine and sulfates. In the final analysis, the pH of precipitation is a measure of the relative contributions of all of these components.

The impact of acidic precipitation on lakes, streams, ponds, forests, fields and manmade objects, therefore, is not the result of a single, or even of several precipitation events, but the result of continued additions of acids or acidifying substances over time. When did precipitation become acidic? Some scientists state that it began with the industrial revolution and the burning of large amounts of coal; others say it began in the United States with the introduction of tall stacks in power plants in the 1950's; other scientists disagree completely and state that rain has always been acidic. In other words, no definitive answer to the question exists at the present time, nor is there data to indicate with any accuracy pH trends in precipitation. The pH of rain has not been continuously monitored in the United States for any period of time, so no data exist. In Scandinavia, on the other hand, the pH of rain has been monitored for many years, therefore a determination of the time of origin can be made.

Though acidic precipitation (wet deposition) is usually emphasized, it is not the only process by which acids or acidifying substances are added to bodies of water or to the land. Dry deposition also occurs. During wet deposition substances such as sulfur and nitrogen oxides are scavenged by precipitation (rain and snow) and deposited on the surface of the earth. Dry deposition processes include gravitational sedimentation of particles, impaction of aerosols and the sorption and absorption of gases by objects at the earth's surface or by the soil or water. Gases, particles and solid and liquid aerosols can be removed by both wet and dry deposition. Dew, fog and frost are also involved in the deposition processes but do not strictly fall into the category of wet or dry deposition. Dry deposition processes are not as well understood as wet deposition at the present time, however, all of the deposition processes contribute to the gradual accumulation of acidic or acidifying substances in the environment. In any event, percipitation at the present time is acidic and has been associated with changes in ponds, lakes and streams that are considered by humans to be detrimental to their welfare.

The most visible changes associated with acidic deposition, that is both wet and dry processes, are those observed in the lakes and streams of the Adirondack Mountains in New York State, the Pre-cambrian Shield areas of Canada and in the Scandinavian countries. In these regions the pH of the fresh water bodies has decreased, causing changes in animal and plant populations. The most readily observable has been the decrease in fish populations.

The chemistry of fresh waters is determined primarily by the geological structure (soil system and bedrock) of the lake or stream catchment basin, by the ground cover and by land use. Near coastal areas (up to 100 miles) marine salts also may be important in determining the chemical composition of the stream, river or lake.

Sensitivity of a lake to acidification depends on the acidity of both wet and dry deposition plus the same factors--the soil system of the drainage basin, the canopy effects of the ground cover and the composition of the waterbed bedrock--that determine the chemical composition of fresh water bodies. The capability, however, of a lake and its drainage basin to neutralize incoming acidic substances is determined largely by the composition of the bedrocks.

Soft water lakes, those most sensitive to additions of acidic substances, are usually found in areas with igneous bedrock which contributes few solids to the surface waters, whereas hard waters contain large concentrations of alkaline earths (chiefly bicarbonates of calcium and sometimes magnesium) derived from limestones and calcareous sandstones in the drainage basin. Alkalinity is associated with the increased capacity of lakes to neutralize or buffer the incoming acids. The extent to which acidic precipitation contributes to the acidification process has yet to be determined.

The disappearance of fish populations from freshwater lakes and streams is usually one of the most readily observable signs of lake acidification. Death of fish in acidified waters has been attributed to the modification of a number of physiological processes by a change in pH. Two patterns of pH change have been observed. The first involves a sudden short-term drop in pH and the second, a gradual decrease in pH with time. Sudden short-term drops in pH often result from a winter thaw or the melting of the snow pack in early spring and the release of the acidic constituents of the snow into the water. Fish may be killed at pH levels above those normally causing death.

A gradual decrease in pH, particularly below 5, can interfere with reproduction and spawning of fish until elimination of the population occurs. In some lakes, aluminum mobilization in fresh waters at a pH below 5 has resulted in fish mortality and appears to be as important as pH.

Although the disappearance of and/or reductions in fish populations are usually emphasized as significant results of lake and stream acidification, changes of equal or greater importance are the effects on other aquatic organisms ranging from waterfowl to bacteria. Organisms at all tropic (feeding) levels in the food web appear to be affected. Species reduction in number and diversity may occur, biomass (total number of living organisms in a given volume of water) may be altered and processes such as primary production and decomposition impaired.

Primary production and decomposition are the bases of the two major food webs (grazing and detrital) within an ecosystem by which energy is passed along from one organism to another through a series of steps of eating and being eaten. Green plants, through the process of photosynthesis, are the primary energy producers in the grazing web, while bacteria initiate the detrital food web by feeding on dead organic matter. Disruption of either of these two food webs results in a decrease in the supply of minerals and nutrients, interferes with their cycling and also reduces energy flow within the affected ecosystems. Acidification of lakes and streams affects both these processes when alteration of the species composition and structure of the pondweed and algae plant communities occurs due to a slowing down in the rate of microbial decomposition.

At present there are no documented observations or measurements of changes in natural terrestrial ecosystems that can be directly attributed to acidic precipitation. The information available is an accumulation of the results of a wide variety of controlled research approaches largely in the laboratory, using in most instances some form of "simulated" acidic rain, frequently dilute sulfuric acid. The simulated "acid rains" have deposited hydrogen (H^+), sulfate ($SO_4^=$) and nitrate (NO_3^-) ions on vegetation and have caused nicrotic lesions in a wide variety of plants species under greenhouse and laboratory conditions. Such results must be interpreted with caution, however, because the growth and morphology of leaves under greenhouse conditions are often not typical of field conditions. Based on laboratory studies, the sensitivity of plants to acidic deposition seems to be associated with the wettability of leaf surfaces. The shorter the time of contact, the lower the resulting dose and the less likelihood of injury.

Soils may become gradually acidified from an influx of hydrogen (H^+) ions. Leaching of the mobilizable forms of mineral nutrients may occur. The rate of leaching is determined by the buffering capacity of the soil and the amount and composition of precipitation. Unless the buffering capacity of the soil is strong and/or the salt content of precipitation is high, leaching will in time result in acidification. At present there are no studies showing this process has occurred because of acidic precipitation.

Damage to monuments and buildings made of stone, and corrosion of metals can result from acidic precipitation. Because sulfur compounds are a dominant component of acidic precipitation and are deposited during dry deposition also, the effects resulting from the two processes cannot be distinguished. In addition, the deposition of sulfur compounds on stone surfaces provides a medium for microbial growth that can result in deterioration.

Human health effects due to the acidification of lakes and rivers have been postulated. Fish in acidified water may contain toxic metals mobilized due to the acidity of the water. Drinking water may contain toxic metals or leach lead from the pipes bringing water into the homes. Humans eating contaminated fish or drinking contaminated water could become ill. No instances of these effects having occurred have been documented.

Several aspects of the acidic precipitation problem remain subject to debate because existing data are ambiguous or inadequate. Important issues include: (1) the rate at which rainfall is becoming more acidic and the rate at which the problem is becoming geographically more widespread; (2) the quantitative contributions of various acids to the overall acidity of rainfall; (3) the relative extent to which the acidity of rainfall in a region depends on local emissions of nitrogen and sulfur oxides versus emissions transported from distant sources; (4)

the relative importance of changes in total mass emission rates compared to changes in the nature of the emission patterns (ground level versus tall stacks) in contributing to regional acidification of precipitation; and (5) the relative contribution of wet and dry deposition to the acidification of lakes and streams.

11.1.2 Ecosystem Dynamics

The emission of sulfur and nitrogen oxides into the atmosphere, their transformation, transport and deposition, either as acidic precipitation or in dry form, as well as the responses of aquatic and terrestrial ecosystems to acidic deposition are all natural phenomena that have been in existence as long as humans can remember. Environmental problems arise because the natural systems are being overloaded by emissions from the combustion of fossil fuels from anthropogenic sources.

Life on the planet Earth depends on the movement of energy and minerals through the biosphere, that thin layer of life surrounding the earth. The living systems (forest, grasslands, cultivated fields, lakes, rivers, estuaries and oceans) within the biosphere obtain energy from the sun, nutrients from the earth's crust, the lithosphere, gases from the atmosphere and water from the hydrosphere. All of the living systems are interdependent. Energy and nutrients move from one to another. The living systems together with their physical environment, the lithosphere, hydrosphere and atmosphere, make up the ecosystem that is the planet Earth (Billings, 1978; Boughey, 1971; Odum, 1971; Smith, 1980).

Ecosystems are basically energy processing systems "whose components have evolved together over a long period of time. The boundaries of the system are determined by the environment, that is, by what forms of life can be sustained by the environmental conditions of a particular region. Plant and animal populations within the system represent the objects through which the system functions" (Smith, 1980).

Ecosystems are composed of biotic (living) and abiotic (non-living) components. The biotic component consists of: (a) producers, green plants that capture the energy of the sun; (b) consumers that utilize the food stored by the producers for their energy; and (c) the decomposers who break down dead organic matter and convert it into inorganic compounds again. (See Table 11-1). The abiotic components are the soil matrix, sediment, particulate matter, dissolved organic matter and nutrients in aquatic systems, and dead or inactive organic matter in terrestrial systems (See Table 11-1) (Billings, 1978; Boughey, 1971; Smith, 1980).

Ecosystems are open systems. They both receive from and contribute to the environment that surrounds them. The environment contributes gases, nutrients, and energy. Ecosystems utilize these substances and, in turn, make their own contributions to the environment. Energy flows through the system unidirectionally while water, gases and nutrients are usually recycled and fed back into the system. The functioning of ecosystems is greatly influenced by the extent to which the gases and nutrients are fed back into the system. When materials are not returned to an ecosystem through recycling, they must be obtained in another way. The organismal populations are the structural elements of the ecosystem through which energy flows and nutrients are cycled (Smith, 1980; Billings, 1978; Odum, 1971).

TABLE 11-1. COMPOSITION OF ECOSYSTEMS

Component	Description
Biotic (biological):	
Individuals	Plants, animals (man), and microorganisms. These are either producers, consumers, or decomposers.
Producers	Green plants.
Consumers	Herbivores, carnivores.
Decomposers	Macroorganisms (mites, earthworms, millipedes, and slugs) and microorganisms (bacteria and fungi).
Populations	Groups of interbreeding organisms of the same kind, producers, consumers or decomposers, occupying a particular habitat.
Communities	Interacting populations linked together by their responses to a common environment.
Abiotic (physical):	
Energy	Radiation, light, temperature, and heat flow.
Water	Liquid, ice, etc.
Atmosphere	Gases and wind.
Fire	Combustion.
Topography	Surface features.
Geological strata	Soil, a complex system. Nutrients. (Minerals)

Energy from the sun is the driving force in ecosystems. If the sun's energy were cut off all ecosystems would cease to function. The energy of the sun is captured by green plants through the process of photosynthesis and stored in plant tissues. This stored energy is passed along through ecosystems by a series of feeding steps, known as food chains, in which organisms eat and are eaten. Energy flows through ecosystems in two major food chains, the grazing food chain and the detrital food chain. The amount of energy that passes through the two food chains varies from community to community. The detrital food chain is dominant in most terrestrial and shallow-water ecosystems. The grazing food chain may be dominant in deep-water aquatic ecosystems (Smith, 1980). The fundamental processes involved in these two food chains are photosynthesis, the capture of energy from the sun by green plants, and decomposition, the final dissipation of energy and the reduction of organic matter into inorganic nutrients.

In addition to the flow of energy, the existence of the living world depends upon the circulation of nutrients through the ecosystems. Both energy and nutrients move through the ecosystem as organic matter. It is not possible to separate one from the other. Both influence the abundance of organisms, the metabolic rate at which they live and the complexity and structure of the ecosystem (Smith, 1980). Nutrients, unlike energy, after moving from the living to the nonliving return to the living components of the ecosystem in a perpetual cycle. It is through the cycling of nutrients that plants and animals obtain the minerals necessary for their existence.

The gaseous and sedimentary cycles are the two basic types of nutrient or biogeochemical cycles. The gaseous cycles involve carbon, oxygen and nitrogen. Water, also, is sometimes considered as belonging to the gaseous cycle. In the gaseous cycles, the main nutrient reservoirs are the atmosphere and the ocean. In the sedimentary cycle, to which phosphorus belongs, the soil and rocks of the earth's crust are the reservoir. The sulfur cycle is a combination of the two cycles because it has reservoirs in both the atmosphere and the earth's crust.

Nitrogen, sulfur and water cycles are involved in acidic deposition. Nitrogen, through the agency of plants (chiefly legumes and blue green algae), moves from the atmosphere to the soil and back (see Figure 4-1, Chapter 4). Human intrusion into the nitrogen cycles include the addition of nitrogen oxides to the atmosphere and nitrates to aquatic ecosystems. Sulfur enters the atmosphere from volcanic eruptions, from the surface of the ocean, from gases released in the decomposition processes and from the combustion of fossil fuels (see Figure 11-1). Both the nitrogen and sulfur cycles have been overloaded by the combustion of fossil fuels by man. For these cycles to function, an ecosystem must possess a number of structured relationships among its components. By changing the amounts of nitrogen and sulfur moving through the cycles, humans have perturbed or upset the structured relationships that have existed for thousands of years and altered the movement of the elements through the ecosystems. The pathways the elements take through the system depend upon the interaction of the populations and their relationships to each other in terms of eating and being eaten.

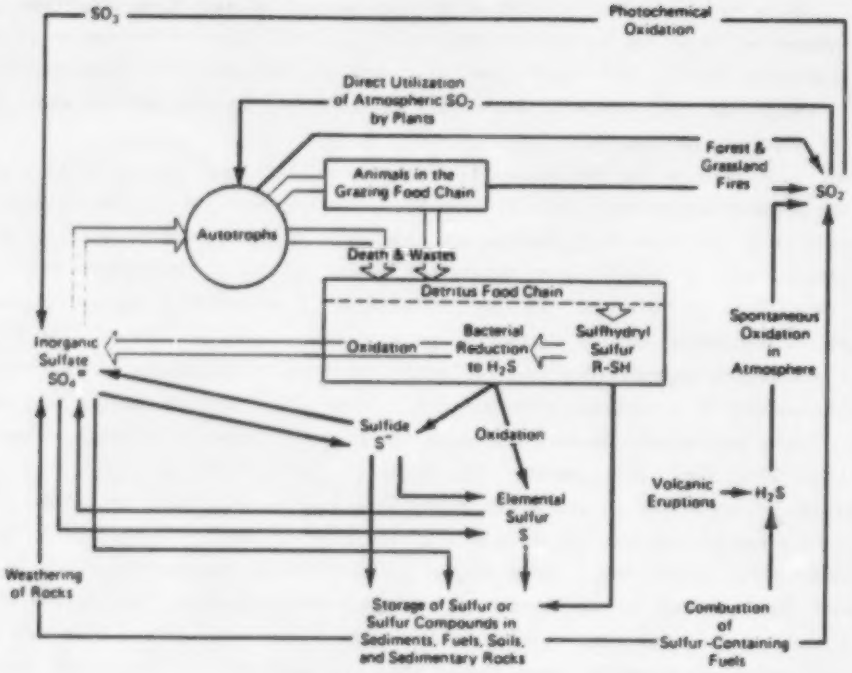

SO₃
Photochemical Oxidation
Direct Utilization of Atmospheric SO₂ by Plants
Forest & Grassland Fires
SO₂
Animals in the Grazing Food Chain
Autotrophs
Death & Wastes
Detritus Food Chain
Inorganic Sulfate SO₄⁼
Oxidation
Bacterial Reduction to H₂S
Sulfhydryl Sulfur R-SH
Spontaneous Oxidation in Atmosphere
Sulfide S⁼
Oxidation
Elemental Sulfur S
Volcanic Eruptions
H₂S
Weathering of Rocks
Storage of Sulfur or Sulfur Compounds in Sediments, Fuels, Soils, and Sedimentary Rocks
Combustion of Sulfur-Containing Fuels

Figure 11-1. The sulfur cycle (organic phase shaded).

Source: Clapham (1973).

Change is one of the basic characteristics of our environment. Weather changes from day to day, temperatures rise and fall, rains come and go, soils erode, volcanoes erupt, and winds blow across the land. These are natural phenomena. Significant environmental changes also result when human beings clear forests, build cities and factories, and dam rivers. All of these environmental changes influence the organisms that live in the ecosystems where the changes are occurring (Moran et al., 1980).

Existing studies indicate that changes occurring within ecosystems, in response to pollution or other disturbances, follow definite patterns that are similar even in different ecosystems. It is, therefore, possible to predict the basic biotic responses of an ecosystem to disturbances such as caused by environmental stress (Garrett, 1967; Odum, 1965; Woodwell, 1962, 1970). These responses to disturbance are (1) removal of sensitive organisms at the species and subspecies level due to differential kill; (2) reduction in the number of plants and animals (standing crop); (3) inhibition of growth or reduction in productivity; (4) disruption of food chains; (5) return to a previous state of development; and (6) modification in the rates of nutrient cycling.

Ecosystems can respond to environmental changes or perturbations only through the response of the populations of organisms of which they are composed (Smith, 1980). Species of organisms sensitive to environmental changes are removed. Therefore, the capacity of an ecosystem to maintain internal stability is determined by the ability of individual organisms to adjust their physiology or behavior. The success with which an organism copes with environmental changes is determined by its ability to produce reproducing offspring. The size and success of a population depends upon the collective ability of organisms to reproduce and maintain their numbers in a particular environment. Those organisms that adjust best contribute most to future generations because they have the greatest number of progeny in the population (Billings, 1978; Odum, 1971; Smith, 1980; Woodwell, 1962, 1970).

The capacity of organisms to withstand injury from weather extremes, pesticides, acidic deposition or polluted air follows the principle of limiting factors (Billings, 1978; Moran et al., 1980; Odum, 1971; Smith 1980). According to this principle, for each physical factor in the environment there exists for each organism a minimum and a maximum limit beyond which no members of a particular species can survive. Either too much or too little of a factor such as heat, light, water, or minerals (even though they are necessary for life) can jeopardize the survival of an individual and in extreme cases a species (Billings, 1978; Boughey, 1971; Moran et al., 1980; Odum, 1971; Smith, 1980). The range of tolerance (see Figure 11-2) of an organism may be broad for one factor and narrow for another. The tolerance limit for each species is determined by its genetic makeup and varies from species to species for the same reason. The range of tolerance also varies depending on the age, stage of growth or growth form of an organism. Limiting factors are, therefore, factors which, when scarce or overabundant, limit the growth, reproduction and/or distribution of an organism (Billings, 1978; Boughey, 1971; Moran et al., 1980; Odum, 1971; Smith, 1980). The increasing acidity of water in lakes and streams is such a factor.

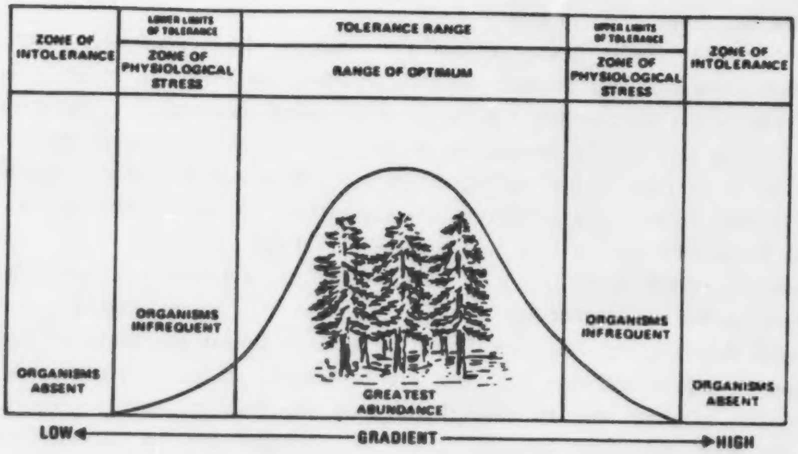

ZONE OF INTOLERANCE
LOWER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
TOLERANCE RANGE
RANGE OF OPTIMUM
UPPER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
ZONE OF INTOLERANCE
ORGANISMS ABSENT
ORGANISMS INFREQUENT
GREATEST ABUNDANCE
ORGANISMS INFREQUENT
ORGANISMS ABSENT
LOW
GRADIENT
HIGH

Figure 11-2. Law of tolerance.

Organisms can exist only within their range of tolerance. Some populations of organisms, annual plants, insects, and mice, for example, respond rapidly. They increase in numbers under favorable conditions and decline rapidly when conditions are unfavorable. Populations of other organisms, such as trees and wolves, fluctuate less in response to favorable or unfavorable conditions. Ecosystems that contain both types of populations are more stable bacause they are able to absorb changes and still persist because the structure of the ecosystem permits it to persist even though populations within it fluctuate widely in response to environmental changes (Holling, 1973; Smith, 1980). Other ecosystems are resistant; their structure enables them to resist changes. Typically, most resistant ecosystems have large living components, trees for example, and store nutrients and energy in the standing biomass. Such resistant systems, such as forests, once highly disturbed are very slow in returning to their original state (Smith, 1980).

Aquatic ecosystems which lack components in which energy and nutrients may be stored for long periods of time usually are not very resistant to environmental changes (Smith, 1980). For example, an influx of pollutants such as effluents from sewage disrupts the system because more nutrients enter the system than it can handle. However, since the nutrients are not retained or recycled within the system it returns to its original state in a relatively short time after the perturbation is removed.

No barriers exist between the various environmental factors or between an organism or biotic community and its environment. Because an ecosystem is a complex of interacting components, if one factor is changed, almost all will change eventually. "The ecosystem reacts as a whole. It is practically impossible to wall off a single factor or organism in nature and control it at will without affecting the rest of the ecosystem. Any change no matter how small is reflected in some way throughout the ecosystem: no 'walls' have yet been discovered that prevent these interactions from taking place" (Billings, 1978).

Continued or severe perturbation of an ecosystem can overcome its resistance or prevent its recovery with the result that the original ecosystem will be replaced by a new system. In the Adirondack Mountains of New York State, in eastern Canada and parts of Scandinavia the original aquatic ecosystems have been and are continuing to be replaced by ecosystems different from the original due to acidification of the aquatic habitat. Forest ecosystems appear to be more resistant because, thus far, changes due to stress from acidifying substances have not been detected. The sections that follow discuss the response of aquatic and terrestrial ecosystems to stressing or perturbation by acidic deposition. Sulfur and nitrogen oxide emissions, their transformation, transport and deposition in acidic form is elucidated in the context of the ecosystem processes that were discussed above.

11.2 CAUSES OF ACIDIC PRECIPITATION

11.2.1 Emissions of Nitrogen and Sulfur Oxides

The generally held hypothesis is that nitrogen and sulfur compounds are largely responsible for the acidity of precipitation (Bolin et al., 1972; Likens and Borman, 1974; Likens, 1976; Smith, 1872). The emissions of the nitrogen and sulfur compounds involved in the acidification are attributed chiefly to the combustion of fossil fuels. Natural sources can also be involved; however, in highly industrialized areas emissions from manmade sources usually exceed those from natural sources (see Chapter 5).

Since 1900 there has been a nearly exponential increase in the consumption of gas, and oil in the United States (see Figure 11-3). Although the total consumption of coal has not increased greatly since about 1925, the consumption of oil and gas has continued to rise precipitously, thus overshadowing coal as the dominant fuel source during the past 50 years (Hubbert, 1976). Within this overall increase in fossil-fuel use, however, there have been shifts in the pattern of consumption. Whereas a considerable proportion of coal was used for transportation and heating, oil and gas have since taken over these functions, and now coal is predominantly devoted to electric power generation (Figure 11-4). In fact, electric power generation is the primary factor accounting for an absolute increase in coal consumption over the past two decades. (The decline in coal use in the 1930s was due to the general economic depression, and the decline in the 1950s was due to the availability of relatively inexpensive oil and gas.) Approximately 550 MM tons (National Research Council, 1978) were used during 1918-1928 compared to 672 MM tons during 1979 (Hamilton, 1980). There was, however, a seasonal shift in the pattern of coal consumption. Summer coal consumption has increased since 1960, while winter consumption has decreased due to increased summer usage by the electric utilities.

These changes in the pattern of fuel use have been accompanied by changes in the pattern of pollutant emissions. Figure 11-5A and 11-5B illustrate the rise since 1940 in emissions of sulfur and nitrogen oxides, the primary gaseous pollutants resulting from the combustion of fossil fuels. Although there has been a net increase in both categories, the more consistent rise has been in emissions of nitrogen oxides. Almost all (93 percent) emissions of sulfur oxides in the United States arise from stationary point sources, principally industrial and power plant stacks. Nitrogen oxide pollutants, on the other hand, originate about equally from transportation (mobile) sources and from stationary sources, which include not only industrial and power plants, but residential and institutional heating equipment as well (U.S. Environmental Protection Agency, 1978). (see Tables 5-2 and 5-3, Chapter 5.)

The geographic distributions of sources of the gaseous precursors of acidic precipitation are depicted in Figures 11-6 and 11-7. Clearly, the dominant sources of sulfur oxides in the United States are in the eastern half of the country, particularly the northeastern quadrant. Major nitrogen oxide sources also show a tendency to be concentrated somewhat in the northeastern quadrant of the country.

Chapter 5, Section 5.2.2 should be consulted for a more detailed account of the sources and emissions of nitrogen oxides.

11.2.2 Transport of Nitrogen and Sulfur Oxides

Among the factors influencing the formation as well as the location where acidic deposition occurs is the long-range transport of nitrogen and sulfur oxides. Neither the gases nor

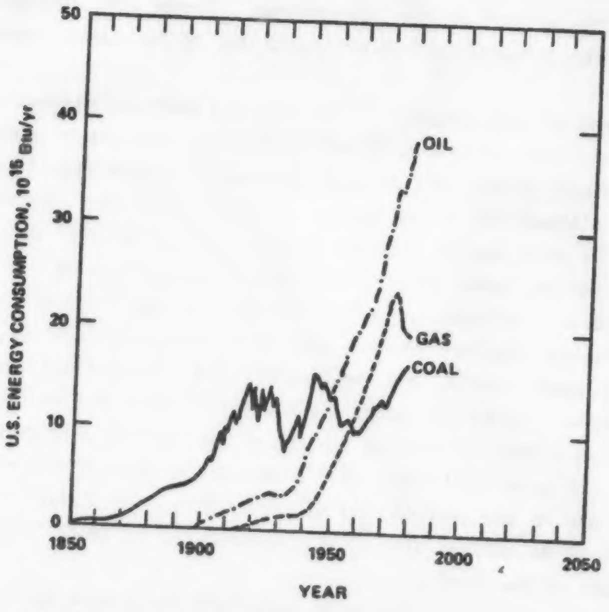


Figure 11-3. Historical patterns of fossil fuel consumption in the United States (adapted from Hubbert, 1976).

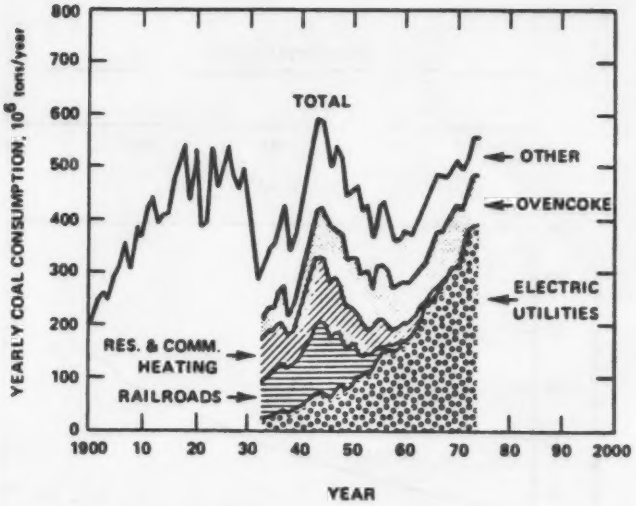
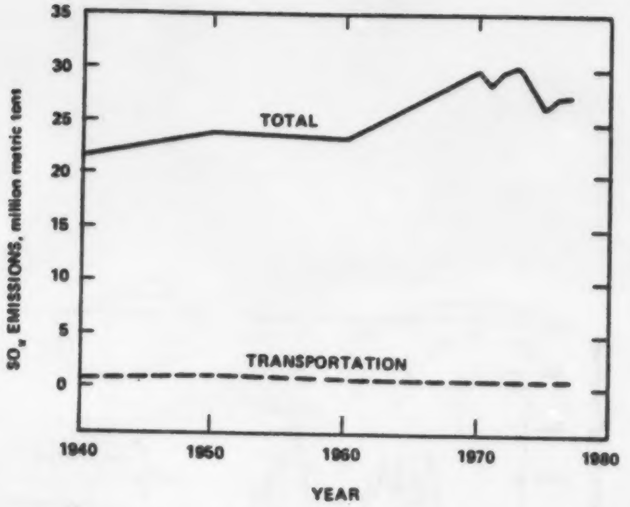


Figure 11-4. Forms of coal usage in the United States. Electric power generation is currently the primary user of coal. (Data from U.S. Bureau of Mines, Minerals Yearbooks 1933-1974)



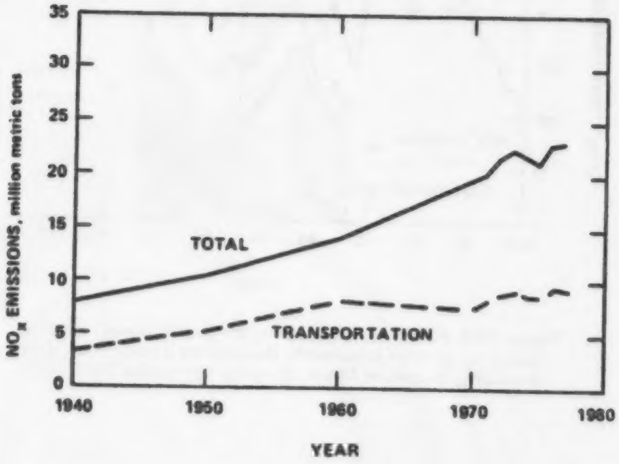


Figure 11-5a. Trends in emissions of sulfur dioxides.

Figure 11-5b. Trends in emissions of nitrogen oxides.

Source: U.S. Environmental Protection Agency (1978).

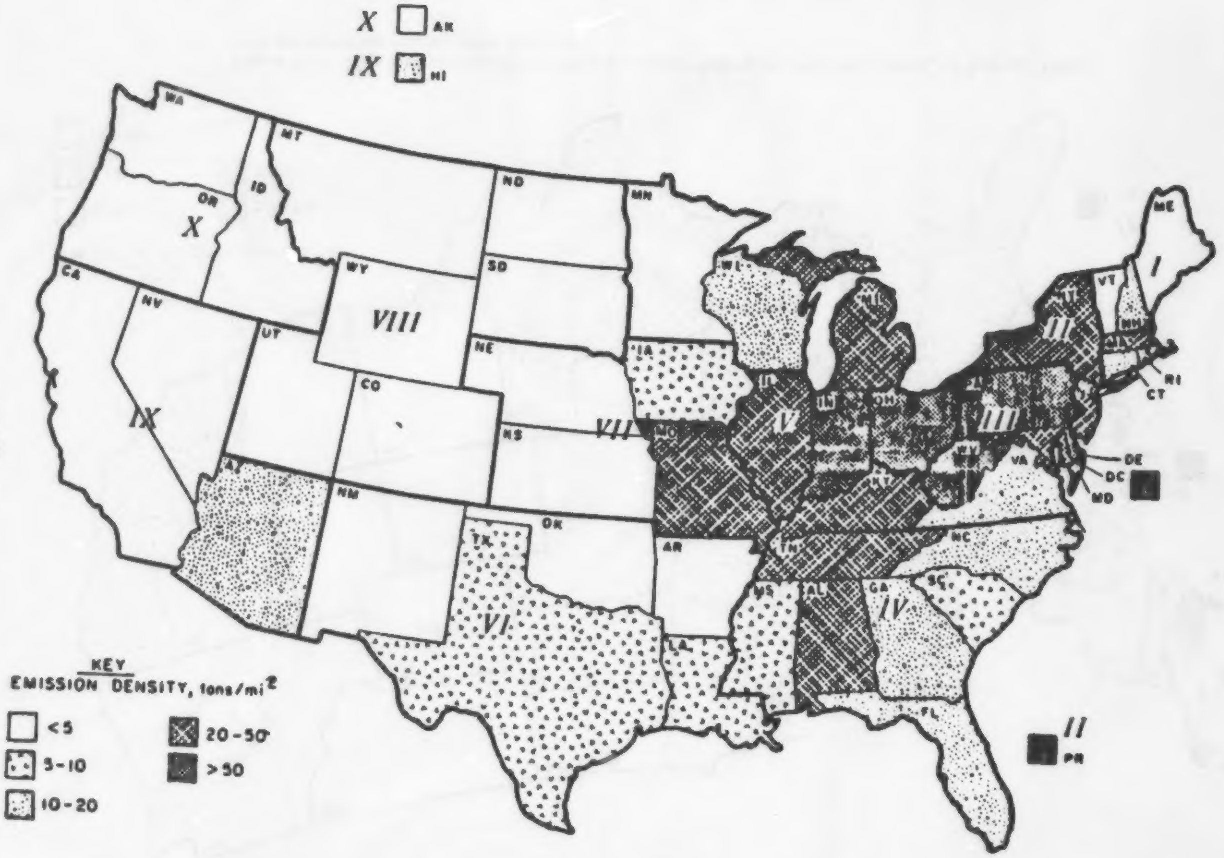
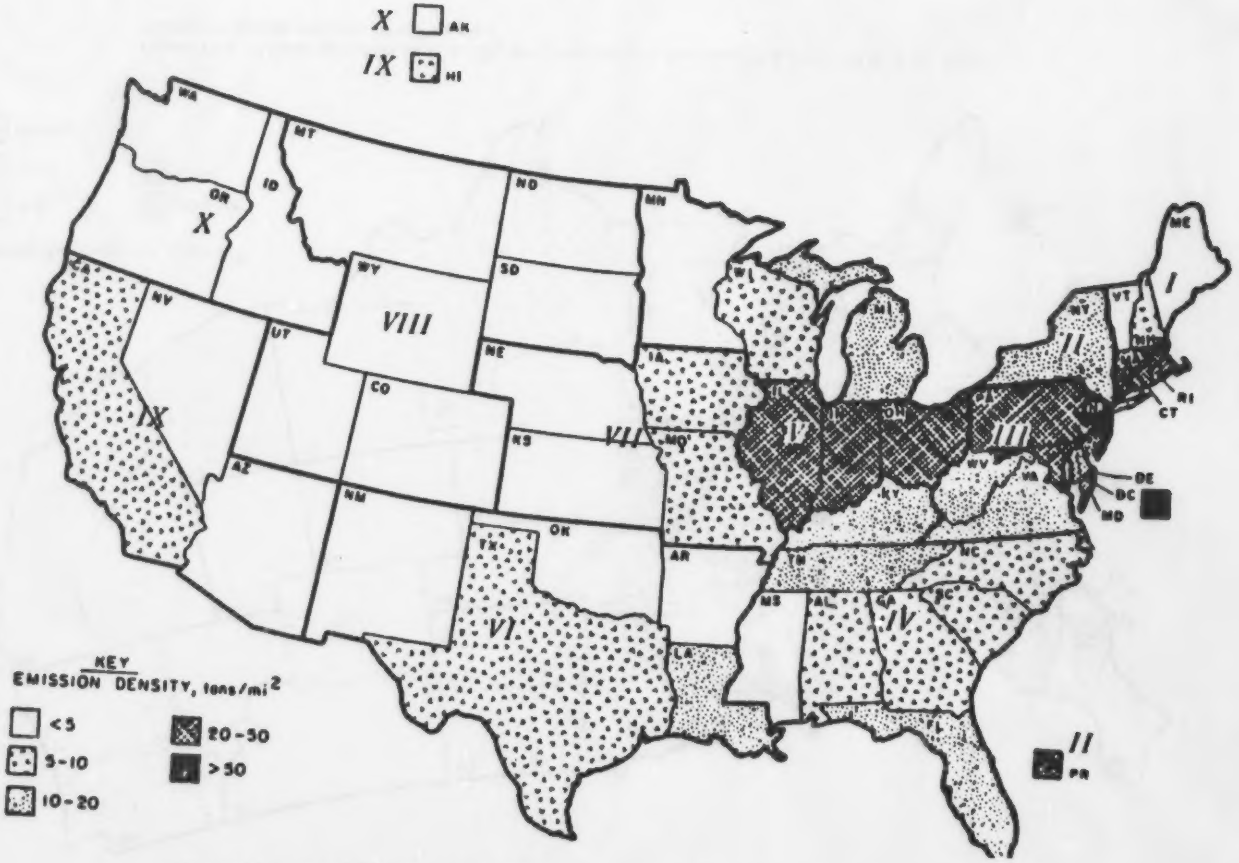

X AK
IX HI
WA
OR
X
CA
NV
IX
ID
MT
WY
VIII
UT
AZ
CO
NM
ND
SD
NE
KS
VII
OK
TX
VI
MN
IA
MO
AR
LA
WI
MI
IL
IN
OH
V
KY
TN
MS
AL
GA
IV
SC
NC
FL
VA
WV
PA
III
NY
II
VT
NH
MA
I
ME
RI
CT
DE
DC
MD
II PR
KEY
EMISSION DENSITY, tons/mi²
<5
5-10
10-20
20-50
>50

Figure 11-6. Characterization of U.S. SO_x emissions density by state (U.S. Dept. of Energy, 1980). (Roman numerals indicate EPA Regions.)


X AK
IX HI
WA
OR
X
ID
MT
ND
MN
CA
NV
UT
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VIII
SD
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CO
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VII
IX
AZ
NM
OK
TX
VI
IA
MO
AR
LA
WI
MI
IL
V
IN
OH
KY
TN
MS
AL
GA
IV
SC
NC
FL
WV
VA
PA
III
NY
II
VT
NH
I
ME
RI
CT
DE
DC
MD
II
PR
KEY
EMISSION DENSITY, tons/mi²
<5
5-10
10-20
20-30
>30

Figure 11-7. Characterization of U.S. NO_x emissions density by state (U.S. Dept. of Energy, 1980). (Roman numerals indicate EPA Regions.)

their transformation products always remain near the sources from which they have been emitted. They may be transported for long distances downwind (Altshuller and McBean, 1979; Cogbill and Likens, 1974; Pack et al., 1978).

The geographic picture of the problem of acidic precipitation in North America can be better understood in the light of some information on prevailing wind patterns. Winds transport the precursors of acidic precipitation from their points of origin to areas where the acidified rain and snow eventually fall. Prevailing winds in the eastern United States tend to be from the west and southwest. Atmospheric pollutants, therefore, are carried in a generally northeasterly direction. Thus, pollution originating in the Ohio River valley can be carried toward the New England states. Seasonal meteorological patterns, however, can modify the direction of windflow, particularly in the summer. The Maritime Tropical air masses from the Gulf of Mexico that occur in late summer have the greatest potential for the formation and transport of high concentrations of sulfate into the northeastern United States and into eastern Canada (Sinclair, 1969).

Cogbill and Likens (1974) associated acidic rainfall in central New York during 1972-73 with high altitude air masses transported into the region from the Midwest. They stated that the NO_x and SO_2 that is involved in acidic rain formation may be transported distances of 300 to 1500 km. Reports by Miller et al., (1978) Wolff et al., (1979) and Galvin et al. (1973) all support the concept that the trajectories of the air masses which come from the Midwest carry sulfur and nitrogen compounds which acidify precipitation in New York State.

A significant though disputed factor in this transport picture is the height at which the pollutants are emitted. Industrial and power plant smokestacks emit their effluents into the atmosphere at higher elevations than do motor vehicles or most space heating equipment. In fact, there has been a trend since the 1960s toward building higher stacks as a means of dispersing pollutants and thereby reducing pollutant concentrations in the vicinity of power plants, smelters, and similar sources (Grennard and Ross, 1974). The result has been that sulfur and nitrogen oxides are carried by prevailing winds for long distances and allowed to diffuse over greater areas through the atmosphere (See Figure 11-8.). Concomitantly, long-range transport allows greater time for chemical reactions to convert these pollutant gases into particulate forms which are more easily removed by wet processes (Eliassen and Saltbones, 1975; Prahm et al., 1976; Smith and Jeffrey, 1975). Chapter 6 discusses the chemical transformations and wet and dry deposition as well as transport and diffusion of nitrogen oxides in the atmosphere. Sulfates and nitrates combine with atmospheric water to form dissociated forms of nitric (HNO_3) and sulfuric (H_2SO_4) acids. These acids are considered to be the main components of acidic precipitation.

The mechanisms of these chemical reactions are quite complex and depend on a host of variables ranging from physical properties of the pollutants to weather conditions and the presence of catalytic or interacting agents (Fisher, 1978). Although these processes of atmospheric chemistry are not well understood, it does appear that the long-range transport of

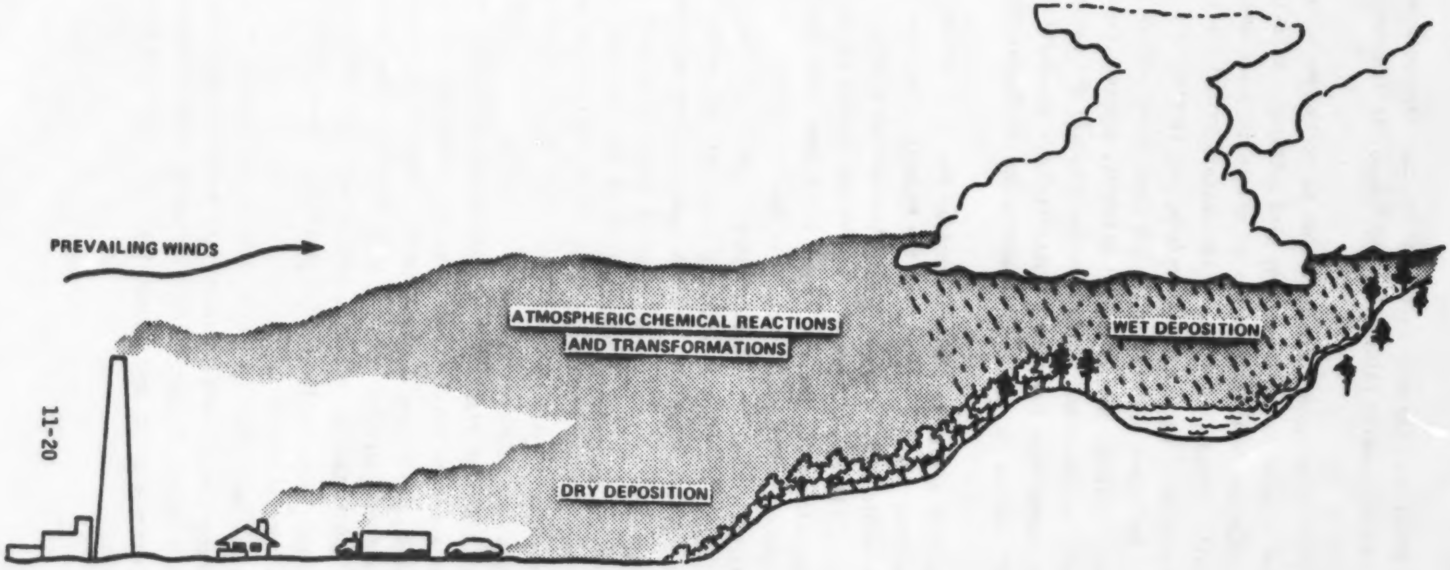

PREVAILING WINDS
ATMOSPHERIC CHEMICAL REACTIONS AND TRANSFORMATIONS
DRY DEPOSITION
WET DEPOSITION

Figure 11-8. The transport and deposition of atmospheric pollutants, particularly oxides of sulfur and nitrogen, that contribute to acidic precipitation.

sulfur compounds can cover 1000 to 2000 km over three to five days (Pack et al., 1978). Thus, the impact of sulfur pollutants in the form of acidic precipitation may be far removed from their points of origin. It is not yet clear whether the atmospheric transport of nitrogen oxide pollutants is comparable to that of sulfur compounds, (Pack, 1978) but in the northeast nitrates are currently thought to contribute 15 to 30 percent of the acidity of polluted precipitation. This figure has increased over the past few years and is expected to increase still further in the future (National Research Council, 1978).

11.2.3 Formation

Precipitation is that portion of the global water cycle by which water vapor from the atmosphere is converted to rain or snow and then is deposited on the earth surfaces (Smith, 1980). Water moves into the atmosphere by evaporation and transpiration (water vapor lost by vegetation). Once it reaches the atmosphere, the water vapor is cooled, then condenses on solid particles and soon reaches equilibrium with atmospheric gases. One of the gases is carbon dioxide. As carbon dioxode dissolves in water, carbonic acid (H_2CO_3) is formed. Carbonic acid is a weak acid and in distilled water only dissociates slightly, yielding hydrogen ions and bicarbonate ions (HCO_3^-). When in equilibrium with normal atmospheric concentrations and pressures of carbon dioxide, the pH of rain and snow is approximately (Likens et al., 1979).

The pH of precipitation may vary and become more basic or more acidic depending on substances in the atmosphere. Dust and debris may be swept from the ground in small amounts and into the atmosphere where it can become a component of rain. Soil particles are usually slightly basic in distilled water and release positive ions, such as calcium (Ca^{2+}), magnesium (Mg^{2+}), potassium (K^+), and sodium (Na^+) into solution. Bicarbonate usually is the corresponding negative ion. Decaying organic matter adds gaseous ammonia to the atmosphere. Ammonia gas in rain or snow forms ammonium ions (NH_4^+) and tends to increase the pH. In coastal areas sea spray plays a strong role in the chemistry of precipitation. The important ions entering into precipitation--sodium, magnesium, calcium, potassium, and the anions chloride (Cl^-) and sulfate (SO_4^{2-})--are also those most abundant in ocean water (Likens, 1976; Likens et al., 1979).

Gases, in addition to CO_2, which enter precipitation, are sulfur dioxide (SO_2) and the nitrogen oxides (NO_x). Some sulfur gases originate from natural sources, e.g. volcanoes and swamps. Others originate from industrial emissions. In the wet atmosphere, both SO_2 and H_2S can be oxidized to sulfuric acid. Nitrogen oxides in the atmosphere are converted to nitric acid (Likens, 1976; Likens et al., 1979). Strong acids dissociate completely in dilute aqueous solutions and lower the pH to less than 5.6. Acidic precipitation has been considered by many scientists to be rain or snow with a pH below 5.6. (Galloway and Cowling, 1978; Likens et al., 1979; Wood, 1975).

Additional acidic or potentially acidifying substances present in both wet and dry deposition are sulfur trioxide ($SO_3^{=}$), sulfate $SO_4^{=}$), nitric oxide (NO), nitrogen dioxide (NO_2), nitrite (NO_2^-), nitrate (NO_3^-), ammonium (NH_4^+), chlorine (Cl^-) hydrochloric acid (HCl), and Brønsted acids [e.g., dissolved iron (Fe) and ammonium (NH_4^+)] (Whelpdale, 1978).

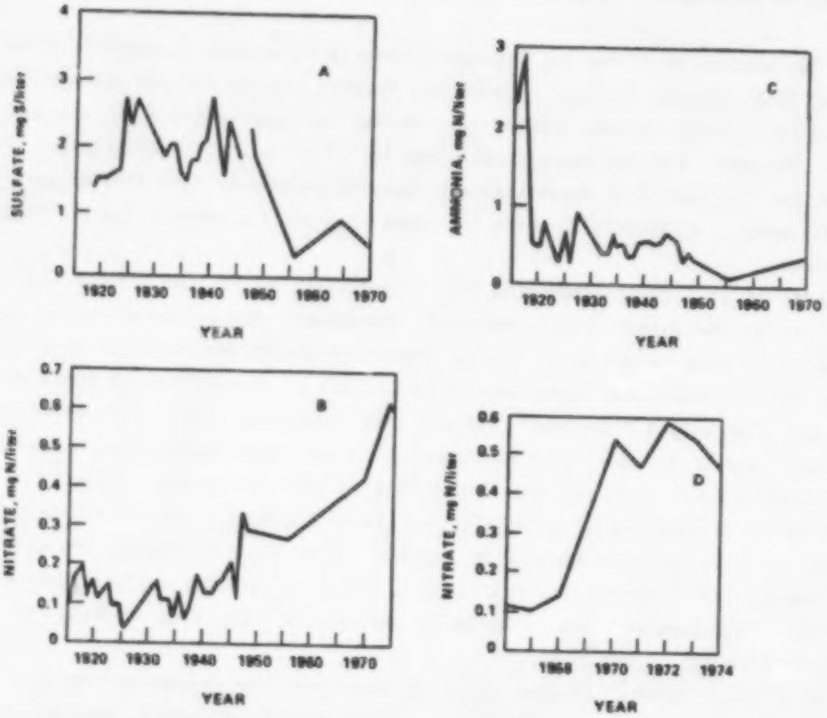
The amounts of the various substances in the atmosphere originating from seawater, desert sands, volcanic islands, or vegetated land influence the chemistry of natural precipitation. In regions with calcareous soils, calcium and bicarbonate may enter precipitation as dust, subsequently increasing the pH of rain or snow to 6.0 or above (Likens et al., 1979).

11.2.3.1 Composition and pH of Precipitation--Sulfur and nitrogen compounds are chiefly responsible for the acidity of precipitation. Continuous measurement of pH in rain by Likens et al., (1972) for the Hubbard Brook Experimental Forest in New Hampshire from 1964 to 1971 indicated the precipitation was acid with an annual weighted average pH range of 4.03 to 4.19. (A weighted average takes into account the amount of rain as well as its composition.) Cogbill and Likens (1974) using precipitation from the Ithaca area, and Hubbard Brook reported that their analysis of precipitation which consistently had a pH of less than 4.4 showed that 65 percent of the acidity was due to H_2SO_4, 30 percent to HNO_3, and less than 5 percent was due to HCl. Hendry (1977) found that sulfate contributed 69 percent, nitrate 23 percent, and chloride 8 percent of the free acidity in rainfall at Gainesville, Florida, during 1976.

In 1976, Likens (1958) reported that the continued monitoring of precipitation at the Hubbard Brook Forest through 1974 indicated the mean annual pH for the years 1964-1974 ranged from 4.03 to 4.21. No statistically significant trend was noted; however, pH values of 2.1 and 3.0 were observed for individual storms at various locations. The increased deposition of hydrogen ion was due to an increase in nitric acid in the precipitation (rain and snow) falling there. This change in the composition of acidic precipitation suggests that the sources of nitrogen oxide emissions increased while those for sulfur oxides remained constant.

The acidity of precipitation is a reflection of the free hydrogen ions in precipitation. The contribution of sulfate and nitrate anions has changed with time, and analysis indicates that the nitrate anion makes up an ever-increasing fraction of the total negative ion equivalents. Following the reasoning of Granat (1972), Likens et al. (1976) found [assuming $2H^+$ per SO_4^{2-} ion as in H_2SO_4 or one H^+ ion per SO_4^{2-} as in $(NH_4)_2SO_4$] that the contribution of sulfate to acidity declined from 83 to 66 percent of the total acidity between 1964 to 1974 at Hubbard Brook, and the contribution of nitrate increased from 15 to 30 percent of the total during the same period. Furthermore, increased annual input of H^+ was closely correlated with increased input of nitrate, but there was little correlation between H^+ input and sulfate input.

Data for nitrate, ammonium, and sulfate in rain at Ithaca and Geneva, New York, constitute the longest record of precipitation chemistry in the United States (Likens, 1972). Data are available from 1915 to the present, but long gaps exist in the measurements, especially at the Geneva site. Figures 11-9 (A) to (C) show that marked changes in composition have occurred at Ithaca: a gradual decline in ammonium, an increase in nitrate beginning around 1945, and a marked decrease in sulfate starting between 1945 and 1950. Early data for Ithaca showed higher concentrations of sulfate in winter than in summer, presumably because of greater local burning of coal in winter. Data for 1971 showed the reverse trend, however, with nearly half the annual sulfate input occurring during the months of June to August. Likens (1972) concluded that, despite deficiencies in the historical data and questions



SOURCE: (A), (B), and (C) modified from Likens (1972); (D) modified from Likens (1976).

Figure 11-9. Trends in mean annual concentrations of sulfate, ammonia, and nitrate in precipitation. (A), (B), and (C) present long-term data for Ithaca, New York; (D) presents data for eight years averaged over eight sites in New York and one in Pennsylvania. One point in (A), for 1946-47, is believed to be an anomaly (see Likens, 1972, for discussion).

concerning their reliability, the trends are real and can be explained by changes in fuel consumption patterns, i.e., natural gas began to replace coal for home heating near the time of the shifts in precipitation chemistry. On the basis of United States Geological Survey data for nine stations, Likens (1976) reported a sharp increase in nitrate concentrations in New York state during the past decade [Figure 11-9 (D)].

Galvin and Cline (1978) sampled the snow in a wilderness area of northern New York State. Analysis of the samples by ion chromatography indicated that only nitrate and sulfate were present in appreciable quantities with the concentration of nitrate being larger than sulfate. The absence of chloride in the samples suggests that the source of the nitrate and sulfate was inland.

Data for eastern North America indicate a roughly three-fold increase in nitrate in rainfall since 1955, whereas sulfate in rain has roughly doubled in this period. According to Nisbet (1975), sulfate/nitrate ratios in rainfall averaged about 4 in the eastern United States in 1955-1956, but the average ratio had fallen to about 3 in 1972-1973. Nisbet calculated that the fraction of H^+ deposition attributable to nitrate rose from 19 percent in 1955-1956 to 24 percent in 1972-1973, while the deposition attributable to H_2SO_4 decreased from 80 to 73 percent.

Lindberg et al. (1979) noted that SO_4^{2-} and H^+ were by far the dominant constituents of precipitation at the Walker Branch Watershed, Tennessee. Comparison with the annual average concentration of major elements in rain at the Walker Branch Watershed on an equivalent basis indicated that H^+ constitutes approximately 50 percent of the cationic strength and trace elements account for only 0.2 percent. Sulfate constituted approximately 65 percent of the anionic strength and on an equivalent basis was 3.5 times more concentrated than NO_3^-, the next most abundant anion. The incident precipitation for the 2-year (1976-1977) period was described as "a dilute mineral acid solution", primarily H_2SO_4, at a pH approximating 4.2 and containing relatively minor amounts of various trace salts (Lindberg et al., 1979). In Florida, Hendry (1977) and Hendry and Brezonik (1980) found that sulfate contributed 69 percent, nitrate 23 percent, and chloride 8 percent of the free acidity in rainfall at Gainesville, Florida, during 1976.

Based on most reports, sulfate (SO_4^{2-}) appears to be the predominant anion in acidic precipitation in the Eastern United States. In the west in California, however, nitrate (NO_3^-) seems to predominate. Liljestrand and Morgan (1978) reported that their analyses of acidic rainfall collected from February 1976 to September 1977 in the Pasedena, CA, area showed that the volume-weighted mean pH was 4.0, with nitric acid being 32 percent more important as a source of acidity than sulfuric acid. The major cations present were H^+, NH_4^+, K^+, Ca^{2+} and Mg^{2+} while the major anions were Cl^-, NO_3^- and SO_4^{2-}. McColl and Bush (1978) also noted the strong influence of nitrate on rain in the Berkeley, CA, region. However, they note that in bulk precipitation (wet plus dry fall-out) that sulfate (SO_4^{2-}) constituted 50 percent of the total anions.

Nearly all of the nitrate in rainfall is formed in the atmosphere from NO_x. Little is derived from wind erosion of nitrate salts in soils. Similarly, nearly all of the sulfate in rainfall is formed in the atmosphere from SO_2 (National Research Council, 1978). Thus, all atmospherically derived nitrate and sulfate contribute to the acidification of precipitation, since H^+ is associated stoichiometrically with the formation of each. A second stoichiometric process that affects the acidity of rain is the reaction of nitric and sulfuric acids with ammonia or other alkaline substances (e.g., dust particles) in the atmosphere to form neutral nitrate and sulfate aerosols. To the extent that such neutralization occurs, the acidity of precipitation will be reduced (National Research Council, 1978). However, since much of the ammonium ion reaching soil is converted to nitrate, these neutral salts still have an acidifying effect on the soil.

11.2.3.2 Seasonal Variations in Nitrates and Sulfates--Seasonal fluctuations in composition as well as pH of rainfall have been reported by many workers. In addition, the composition of rainfall and pH fluctuates from event to event, from locality to locality, and from storm to storm.

In general SO_4^{2-} and H^+ concentrations in precipitation in the eastern United States are higher in the summer than in the winter. Wolff et al. (1979) found this to be true for the New York Metropolitan Area. Hornbeck et al. (1976) and Miller et al. (1978) both stated that a summer maximum for sulfate was associated with an increase in hydrogen ion concentration in upstate New York, the Hubbard Brook Experimental Forest in New Hampshire, and in portions of Pennsylvania. Pack (1978) using data (1977) from the four original MAP3S (Multistate Atmospheric Power Production Pollution Study) precipitation chemistry networks, plotted the weighted monthly sulfate ion concentrations (Figure 11-10). Maximum sulfate concentrations occurred from June through August. Lindberg et al. (1979) studying wetfall deposition of sulfate in the Walker Branch Watershed, also noted summer maxima for SO_4^{2-} and H^+. Using the same MAP3S data as did Pack, they plotted weighted mean concentrations of sulfate in rain collected from November 1976 through November 1977. The concentrations at Walker Branch Watershed, Tennessee, are lower than all of the stations except remote Whiteface Mountain, New York. The regional nature of the wet deposition of sulfate is apparent. Reasons for the existence of the high summer maxima of sulfate for the eastern United States are discussed in some detail in Chapter 5, Section 5.3.4.

Seasonal variations of nitrogen compounds and of pH in precipitation have been reported by several workers, but no simple trends are apparent (see U.S. Environmental Protection Agency Air Quality Criteria for Nitrogen Oxides, 1980). Hoeft et al. (1972) found relatively constant levels of nitrate in rain and snow collected in Wisconsin throughout the year, but deposition of ammonia and organic nitrogen was lowest in winter and highest in spring, perhaps because of the thawing of frozen animal wastes. Haines (1976) reported large random variations, but relatively small seasonal variations, for nitrogen forms in wet-only precipitation at Sapelo Island, Georgia; nitrogen concentrations were lowest during the rainy months of July and September. The highest nitrogen loadings occurred during July and were asociated with

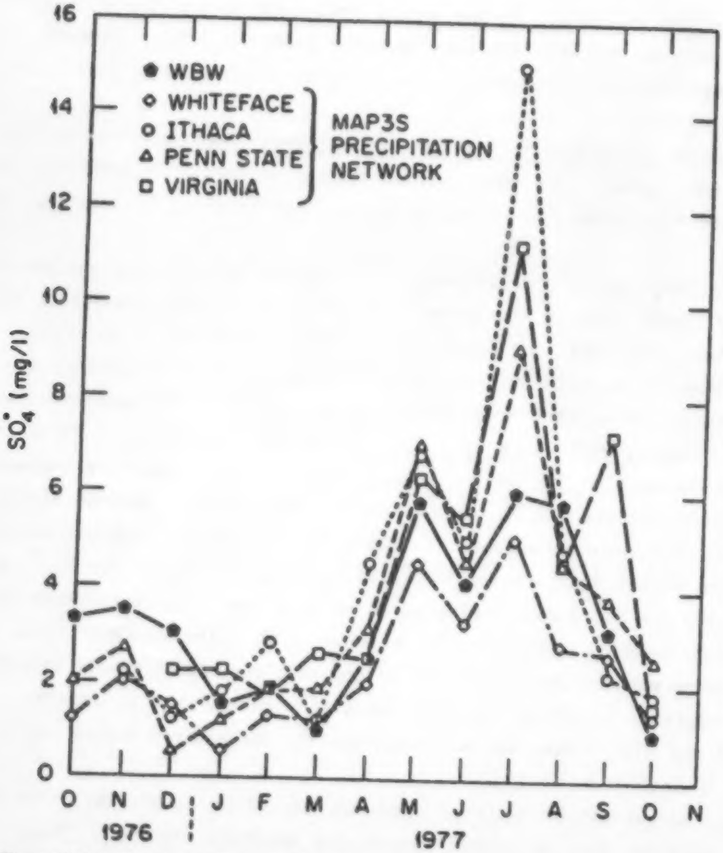


Figure 11-10. Comparison of weighted mean monthly concentrations of sulfate in incident precipitation collected in Walker Branch Watershed, Tenn. (WBW) and four MAP3S precipitation chemistry monitoring stations in New York, Pennsylvania, and Virginia (Lindberg et al., 1979).

the lowest range in pH, 4.2-4.8. Hendry (1977) and Hendry and Brezonik (1980) found relatively smooth seasonal trends in ammonia and nitrate concentrations in both wet-only and bulk collections (wet- and dryfall) at Gainesville, Florida, with lowest concentrations in winter (Figure 11-11). In addition, the pH of the bulk precipitation showed no seasonal trend. Wet-only collections, however, showed the lowest pH value (4.0) during the spring and summer. This historical record suggests there has been an increase in the concentration of inorganic nitrogen in Florida over the past 20 years.

Scavenging by rainfall produces large changes in atmospheric contaminant concentrations during a given rainfall event. The decline in constituent levels is usually rapid, at least in localized convective showers, and low, steady-state concentrations are usually reached within the first half hour of a rain event.

Major ions [chloride (Cl^-) and sulfate ($SO_4^=$)], inorganic forms of nitrogen [nitrate (NO_3^-) and ammonium (NH_4^+)], total phosphorus and pH were measured in rain collected in 5-minute segments within three individual rainstorms. Initially, rapid decreases were observed for nitrate and ammonium and total phosphorus. There was also a decrease in pH from 4.65 to 4.4. Steady state concentrations were reached in 10 minutes. Two other storms sampled in the same manner showed similar but less defined patterns (Hendrey and Brezonik, 1980).

Wolff et al. (1979) examined spatial meteorological and seasonal factors associated with the pH of precipitation in the New York Metropolitan Area. Seventy-two events were studied rom 1975 through 1977. There was some site-to-site variability among the eight sites they studied in the Manhattan area (Table 11-2). They also noted that the pH varied according to storm type (Table 11-3). Storms with a continental origin have a lower pH than storms originating over the ocean. The storms with trajectories from the south and southwest had the lowest pH's, while those from the north and east had the highest pH's (Wolff et al., 1979).

The mean pH of precipitation falling on the New York Metropolitan Area during a 2-year (1975 to 1977) study was 4.28; however, a pronounced seasonal variation was observed (Figure 11-12). The minimum pH at all sites except Manhattan occurred during July to September, while the maximum occurred during October to December. The minimum pH in Manhattan, however, occurred January to March and then gradually increased through the year. The lowest pH of 4.12 for the New York Metropolitan area occurred during the summer months (Wolff et al., 1979). In general, the pH of rain is usually lower in the summer than in the winter and is associated with the high summertime sulfate concentrations. In addition, the lowest pH's were associated with cold fronts and air mass type precipitation events. These events occur more frequently during the summer months. The lower pH's also occurred on westerly or southwesterly winds (Wolff et al., 1979).

Seasonal variations in pH measured at several sites in New York State 70 km (45 mi.) apart demonstrated a significant difference between seasons (Winter had an average pH of 4.2; summer, 3.9.) but no significant difference between sites. In New Hampshire, however, six

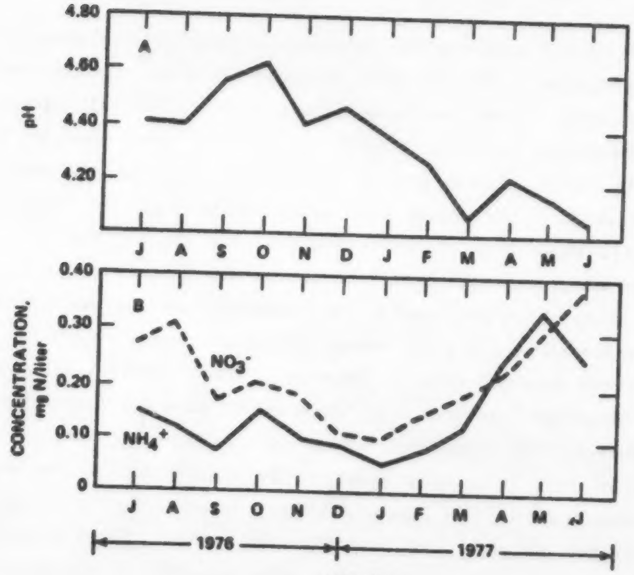


Figure 11-11. Seasonal variations in pH (A) and ammonium and nitrate concentrations (B) in wet-only precipitation at Gainesville, Florida. Values are monthly volume-weighted averages of levels in rain from individual storms (Hendry, 1977).

TABLE 11-2. MEAN pH VALUES IN THE NEW YORK METROPOLITAN AREA (1975-1977)

Site	Mean pH	SD	No. obsd	Range
Caldwell, N.J.	4.32	0.26	50	3.35-5.60
Piscataway, N.J.	4.25	0.36	64	3.57-5.50
Cranford, N.J.	4.34	0.34	48	3.44-5.95
Bronx, N.Y.	4.31	0.37	57	3.42-5.75
Manhattan, N.Y.	4.29	0.25	39	3.80-5.50
High Point, N.J.	4.25	0.30	25	3.74-4.90
Queens, N.Y.	4.63	0.35	20	3.98-5.28
Port Chester, N.Y.	4.60	0.19	21	4.00-5.10
All sites	4.28	0.32	72	3.50-5.16

From Wolff et al. (1979).

TABLE 11-3. STORM TYPE CLASSIFICATION

Type	Description of dominant storm system	No. obsd	Mean pH
1	Closed low-pressure system which formed over continental N. Amer.	22	4.35
2	Closed low-pressure system which formed in Gulf of Mexico or over Atlantic Ocean	21	4.43
3	Closed low which passed to W or N of N.Y.C.	26	4.39
4	Closed low which passed to S or E of N.Y.C.	17	4.39
5	Cold front in absence of closed low	16	4.17
6	Air mass thunderstorm	5	3.91
7	Hurricane Belle	1	5.16
8	Unclassified	6	4.31

From Wolff et al. (1979).

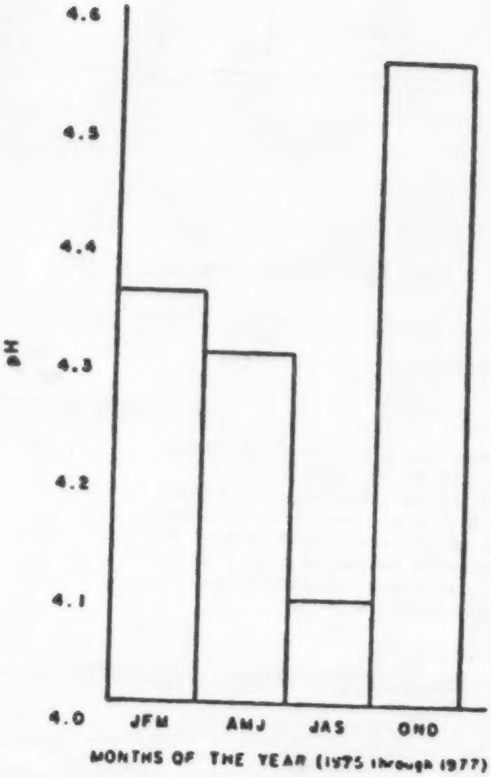


Figure 11-12. Seasonal variation of precipitation pH in the New York Metropolitan Area (Wolff et al, 1979).

summer storms sampled at 4 sites less than 3 km (2 mi.) apart showed a significant difference (3.8 to 4.2) indicating considerable variation in pH may occur in the same storm.

Stensland (1978, 1980) compared the precipitation chemistry for 1954 and 1977 at a site in central Illinois. The pH for the 1954 samples had not been measured, but were calculated and compared with those measured in 1977. The corrected pH for 1954 was 6.05; the pH for 1977 was 4.1. The more basic pH in 1954, according to the author, could have resulted from low levels of acidic ions (e.g. sulfate or nitrate) or from high amounts of basic ions (e.g. calcium and magnesium). Stensland suggests that the higher pH in 1954 was due to calcium (Ca^{++}) and magnesium (Mg^{++}) ions from the soil.

11.2.3.3 Geographic Extent of Acidic Precipitation--Acidic precipitation has been a reality in New York State for an undetermined period of time. Data collected by the United States Geological Survey (Harr and Coffey, 1975) over a ten-year period are presented in Figure 11-13. These curves represent the pH of precipitation at eight different locations in New York State and one location in Pennsylvania. Each of these locations represents an area within a given watershed. The pH of precipitation has remained nearly at the same general average during the entire ten-year period; therefore, since data for the years prior to 1965 are lacking, it is difficult to determine when the pH in precipitation first began to decrease (Harr and Coffey, 1975).

That precipitation is acidic in parts of the country other than the northeastern United States is apparent (see Figure 11-14). Average pH values around 4.5 have been reported as far south as northern Florida (Hendrey and Brezonik, 1980; Likens, 1976), from Illinois (Irving, 1978), the Denver area of Colorado (Lewis and Grant, 1980) the San Francisco Bay area of California (McColl and Bush, 1978; Williams, 1978), Pasadena, California (Liljestrand and Morgan, 1978), the Puget Sound area of Washington (Larsen et al., 1976), and from eastern Canada (Dillon et al., 1978; Glass et al., 1979). Data from the San Francisco Bay area indicate that precipitation has be come more acidic in that region since 1957-1958 (McColl and Bush, 1978). The pH decreased from 5.9 during 1957-1958 to 4.0 in 1974, and seems to be related to an increase in the NO_3^- concentration (McColl and Bush, 1978). Another report, using data from the California Air Resources Board (CARB) (Williams, 1978), states that acidic precipitation has been reported from such widespread areas as Pasadena, Palo Alto, Davis, and Lake Tahoe.

Studies in the Great Smoky Mountain National Park (Lipske, 1980) indicate a downward trend in pH has occurred there over the past twenty years. Over a period of 20 years, there has been a drop in pH from a range of 5.3-5.6 to 4.3 in 1979.

The absence of a precipitation monitoring network throughout the United States in the past makes determination of trends in pH extremely difficult and controversial. This shortcoming has been rectified recently through the establishment of the National Atmospheric Deposition Program funded by State, Federal and private agencies and headquartered at North Carolina State University, Raleigh, N.C. Under the program, monitoring stations collect

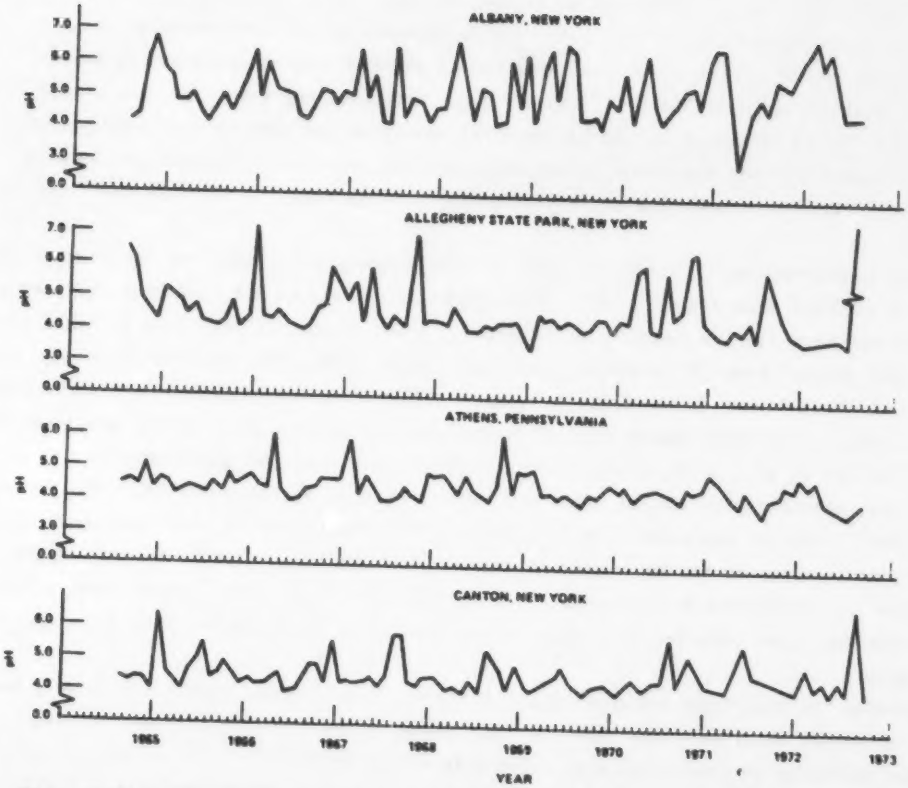


Figure 11-13. History of acidic precipitation at various sites in and adjacent to State of New York (Harr and Coffey, 1975).

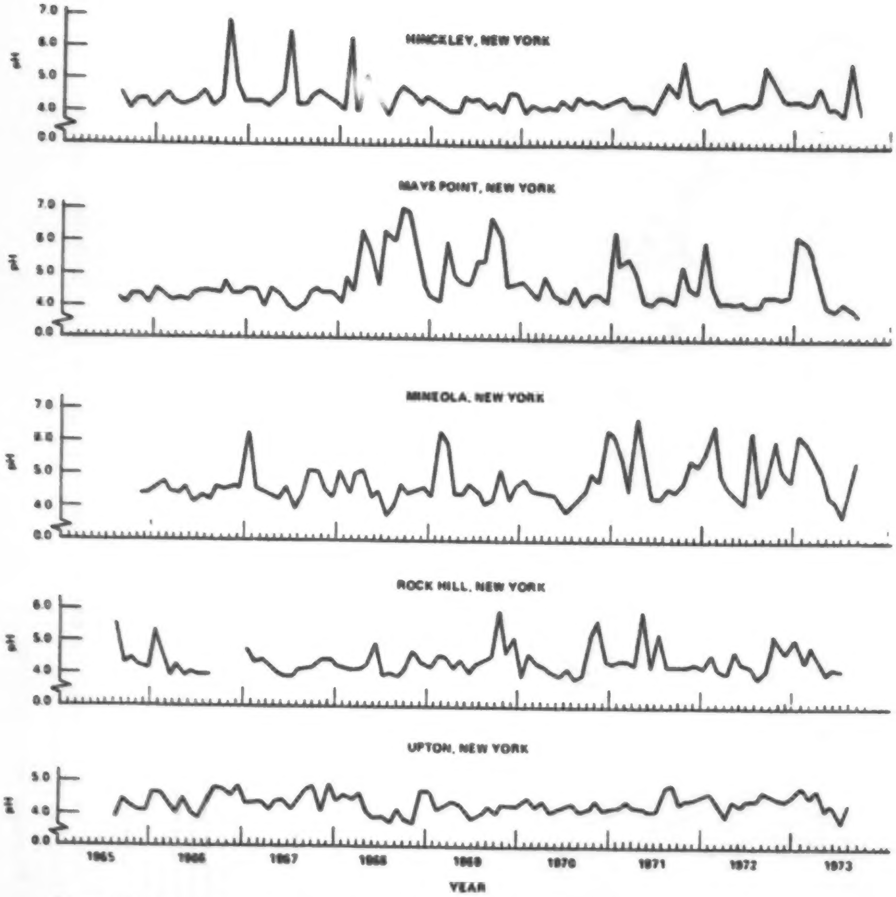


Figure 11-13 (cont'd). History of acidic precipitation at various sites in and adjacent to State of New York (Harr and Coffey, 1975).

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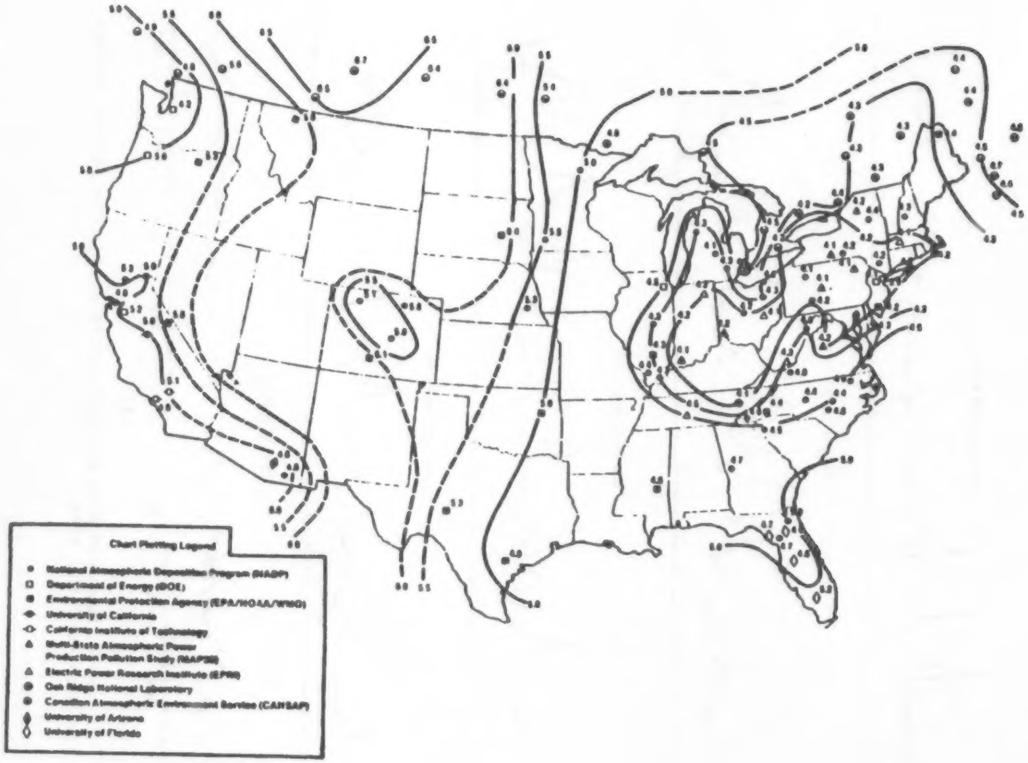
Chart Plotting Legend
● National Atmospheric Deposition Program (NADP)
□ Department of Energy (DOE)
■ Environmental Protection Agency (EPA/NOAA/WMO)
◆ University of California
◇ California Institute of Technology
▲ Multi-State Atmospheric Power Production Pollution Study (MAP3S)
△ Electric Power Research Institute (EPRI)
● Oak Ridge National Laboratory
● Canadian Atmospheric Environment Service (CANSAP)
♦ University of Arizona
◇ University of Florida

Figure 11-14. pH of rain samples as measured in the laboratory and used in combination with the reported amount of precipitation.

Source: Wisniewski and Keitz (1981).

precipitation samples, determine their pH and then send the samples to a Central Analytical Laboratory in Illinois to be analyzed. This long-term network plans to have 75 to 100 collection sites throughout the United States; 74 are already operational.

11.2.4 Acidic Deposition

The previous sections of this chapter have discussed the formation, composition and geographic distribution of acidic precipitation. Usually when the effects of acidic deposition are discussed, emphasis is placed on the effects resulting from the scavenging of sulfur and nitrogen compounds by precipitation. Dry deposition of gaseous and particulate and aerosol forms of these compounds also occurs and is beginning to receive more emphasis in research (Chamberlain, 1980; Galloway and Wheledale, 1980; Schlesinger and Hasey, 1980; Schmel, 1980; Stensland, 1980). Gaseous compounds reach the surface of the earth by turbulent transfer while particulate sulfates and nitrates reach the earth's surface by gravitational sedimentation, turbulent transfer and impaction (Galloway and Whelpdale, 1980; Hicks and Wesely, 1980; Schmel, 1980). A comparison of the relative significance of wet and dry deposition is difficult. Dry deposition, however, is always removing pollutants from the atmosphere, while removal by wet deposition is intermittent (Schmel, 1980). Marenco and Fontan (1974) suggest that dry deposition is more important than wet in removing air pollutants from manmade sources.

Lindberg et al. (1979) have calculated the annual mass transfer rates of sulfates to the forest floor in Tennessee (Figure 11-15). Their calculations for $SO_4^=$ suggest wet deposition by incident precipitation to be 27 percent compared with a total dry deposition of 13 percent. The dry deposition and foliar absorption of SO_2, a very important component, is missing from this calculation. The wet and dry deposition percentages are only an indication of the relative magnitude of the two processes. The percentages do, however, point out that the effects of acidic deposition usually attributed to precipitation scavenging alone are probably a result of both wet and dry deposition. At the present time the accuracy with which dry deposition can be measured is still under question.

The studies of McColl and Bush (1978), Hendry and Brezonik (1980), and Schlesinger and Hasey (1980) also point out that both wet and dry deposition are important when considering the effects of H^+, SO_4^{2-}, and NO_3^- ions on aquatic and terrestrial receptors.

The effects of the dry deposition of SO_2 and particulate matter on vegetation and terrestrial ecosystems is discussed in Chapter 8. The processes of wet and dry deposition of sulfur oxides are discussed in Chapter 6 of Air Quality Criteria for Particulate Matter and Sulfur Oxides. The nitrogen cycle is discussed in Chapter 4, transformation and transport of nitrogen oxides in Chapter 6 and the effects of nitrogen oxides on vegetation in Chapter 12 of this document.

11.3 EFFECTS OF ACIDIC DEPOSITION

Acidic precipitation has been implicated in the degradation of aquatic ecosystems, the disintegration of stone buildings and monuments and as a potential source of harm to forests and other terrestrial ecosystems. The sections that follow discuss these effects.

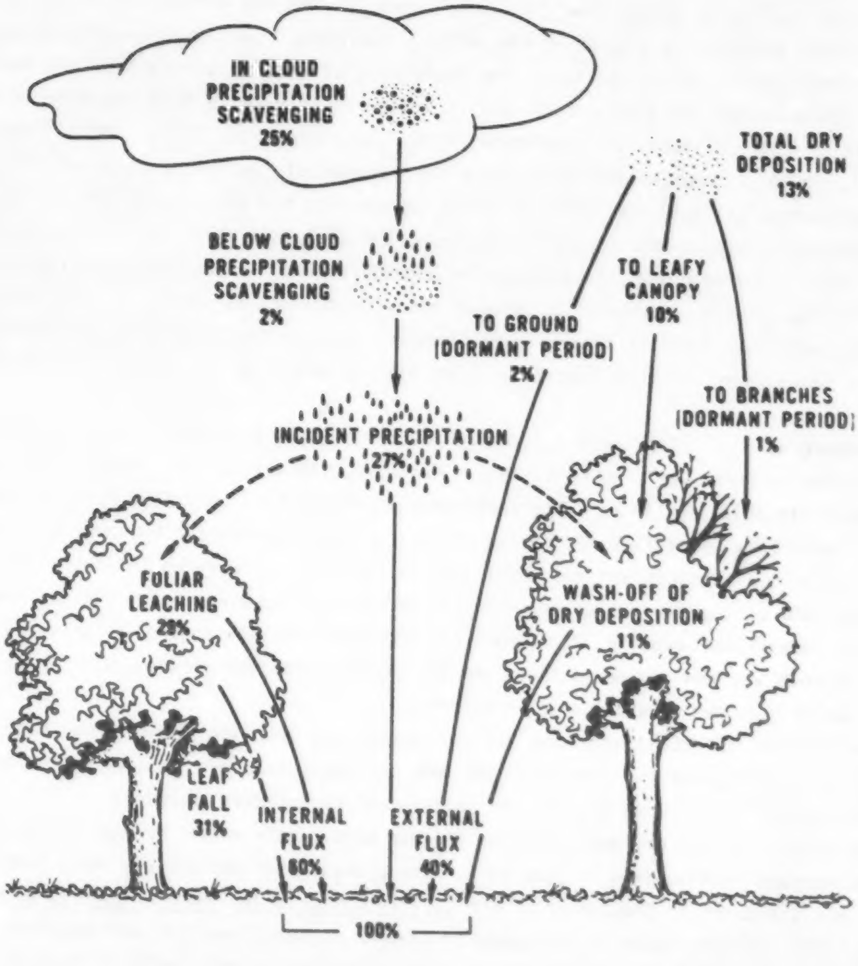
IN CLOUD
PRECIPITATION
SCAVENGING
25%
TOTAL DRY
DEPOSITION
13%
BELOW CLOUD
PRECIPITATION
SCAVENGING
2%
TO LEAFY
CANOPY
10%
TO GROUND
(DORMANT PERIOD)
2%
TO BRANCHES
(DORMANT PERIOD)
1%
INCIDENT PRECIPITATION
27%
FOLIAR
LEACHING
29%
WASH-OFF OF
DRY DEPOSITION
11%
LEAF
FALL
31%
INTERNAL
FLUX
60%
EXTERNAL
FLUX
40%
100%

Figure 11-15. Annual mass transfer rates of sulfate expressed as a percentage of the estimated total annual flux of the element to the forest floor beneath a representative chestnut oak stand (Lindberg et al., 1979).

11.3.1 Aquatic Ecosystems

Acidification of surface waters is a major problem in regions of southern Scandinavia (Almer et al., 1974; Gjessing et al., 1976; Oden, 1968), Scotland (Wright et al., 1980), eastern Canada (Beamish and Harvey, 1972; Dillon et al., 1978), and the eastern United States - in the Adirondack Region of New York State (Pfeiffer and Festa, 1980; Schofield, 1976), in Maine (Davis et al., 1978), and in northern Florida (Crisman et al., 1980). Damage to fisheries is the most obvious affect of acidification on freshwater life. The disappearance of fish populations from acidified freshwater lakes and streams was first noted in southern Norway in the 1920's. In 1959, Dannevig (1976) proposed that acidic deposition was the probable cause for acidification and thus far the loss of fish populations (Lievestad et al., 1976). Subsequent studies have verified this postulate. Declines in fish populations have been related to acidification of surface waters in southern Norway (Jensen and Snekvik, 1972; Wright and Snekvik, 1978), southwestern Sweden (Almer et al., 1974) southwestern Scotland (Wright et al., 1980a), the Adirondack Region of New York State (Schofield, 1976), and the LaCloche Mountain Region in southern Ontario (Beamish and Harvey, 1972). Acidification may also have serious repercussions on other aquatic biota inhabiting these systems. Changes in the acidity and chemistry of freshwater affect the communities of organisms living there. Pertinent details of these effects are described in the following sections.

11.3.1.1 Acidification of Lakes and Streams--Precipitation enters lakes directly as rain or snow or indirectly as runoff of seepage water from the surrounding watershed. The relative magnitude of the influents from these two sources is dependent on the surface area and volume of the lake, and the size of the watershed and its soil volume and type. In general, the watershed plays a dominant role in determining the composition of water entering the lake. As a result, the water will be strongly influenced by processes in the edaphic environment of the watershed, such as weathering, ion exchange, uptake and release of ions by plants, carbon dioxide production by vegetation, microbial respiration, and reduction and oxidation reactions of sulfur and nitrogen compounds (Seip, 1980). Precipitation as a direct source of water to the lake plays a relatively greater role when lake areas are large in comparison to the size of the watershed.

Acidification of surface waters results when the sources of hydrogen ion exceed the ability of an ecosystem to neutralize the hydrogen ion. In general, the soils and crust of the earth are composed principally of basic materials with large capacities to buffer acids. However, areas where bedrock is particularly resistant to weathering and soils are thin and poorly developed have much less neutralizing ability. This inability to neutralize hydrogen ion does not arise from a limited soil or mineral buffering capacity. Instead low cation exchange capacity and slow mineral dissolution rates in relation to the relatively short retention time of water within the soil system may result in incomplete neutralization of soil waters and acidification of surface waters (Driscoll, 1980). Characteristics of regions sensitive to surface water acidification are discussed in more detail in Section 11.4.1.

Sources of hydrogen ion to the edaphic-aquatic system include, besides acidic deposition, mechanisms for internal generation of hydrogen ion - oxidation reactions (e.g., pyrite oxidation, nutrification), cation uptake by vegetation (e.g., uptake of NH_4^+ or Ca^{++}), or generation of organic acids from incomplete organic litter decomposition (Figure 11-16). The relative importance of the hydrogen ion content in acidic deposition to the overall hydrogen ion budget of an ecosystem has been discussed by many researchers (Rosenquist, 1976; SNSF Project, 1977).

The consensus is that changes in internal hydrogen ion generation related to land use or other changes (e.g., Drablos and Sevaldrud, 1980) can not consistently account for the widespread acidification of surface waters occurring in southern Scandinavia, the Adirondack Region of New York, the LaCloche Mountain Area of Ontario, and elsewhere. Driscoll (1980) developed a hydrogen ion budget for the Hubbard Brook Area in New Hampshire. Based on these calculations, atmospheric hydrogen ion sources represent 48 percent of the total Hubbard Brook ecosystem hydrogen ion sources.

As noted above, freshwater ecosystem sensitive to inputs of acids are generally in areas of poor soil development and underlain by highly siliceous types of bedrock resistant to dissolution through weathering (Likens et al, 1979). As a result, surface waters in such areas typically contain very low concentrations of ions derived from weathering. The waters are diluted with low levels of dissolved salts and inorganic carbon, and low in acid neutralizing capacity. The chemical composition of acid lakes is summarized in Table 11-4 for lakes in southern Norway (Gjessing et al., 1976), the west coast (Hornström et al., 1976), and west-central regions of Sweden (Grahn et al., 1976), the LaCloche Mountains of southeastern Ontario (Beamish, 1976), and the vicinity of Sudbury, Ontario (Scheider et al., 1976), as well as for lakes not yet affected by acidification but in regions of similar geological substrata in west-central Norway (Gjessing et al., 1976) and the experimental lakes area of northwestern Ontario (Armstrong and Schindler, 1971). Basic cation concentrations (Ca, Mg, Na, K) are low (e.g., calcium levels of 18-450 μeq/liter or 0.4 - 9 mg/liter) relative to world-wide averages [15 mg/liter calcium (Livingstone, 1963)]. Bicarbonate is the predominant anion in most freshwaters (Stumm and Morgan, 1970). However, in acid lakes in regions affected by acidic deposition, sulfate replaces bicarbonate as the dominant anion (Beamish, 1976; Wright and Gjessing, 1976). With a decreasing pH level in acid lakes, the importance of the hydrogen ion to the total cation content increases.

Surveys to determine the extent of effects of acidic deposition on the chemistry of lakes have been conducted in Norway (Wright and Snekvik, 1976; Wright and Henriksen, 1978), Sweden (Almer et al., 1974; Dickson, 1975), Scotland (Wright et al., 1980a), the LaCloche Mountain area of Ontario (Beamish and Harvey, 1972), the Muskoka-Haliburton Area of south-central Ontario (Dillon et al., 1978), and the Adirondack Region of New York State (Schofield, 1976b), Maine (Davis et al., 1978), and Pennsylvania (Arnold et al., 1980). In regions of similar geological suostrata not receiving acidic deposition, lake pH levels average 5.6-6.7 (Armstrong and Schindler, 1971). Of 155 lakes systematically surveyed in southern Norway in

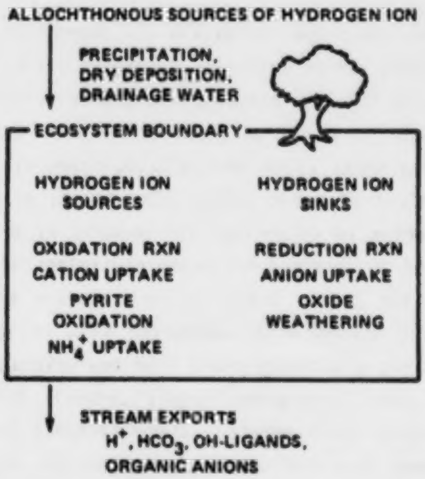
ALLOCHTHONOUS SOURCES OF HYDROGEN ION
PRECIPITATION,
DRY DEPOSITION,
DRAINAGE WATER
ECOSYSTEM BOUNDARY
HYDROGEN ION SOURCES
HYDROGEN ION SINKS
OXIDATION RXN
REDUCTION RXN
CATION UPTAKE
ANION UPTAKE
PYRITE OXIDATION
OXIDE WEATHERING
NH_4^+ UPTAKE
STREAM EXPORTS
H^+, HCO_3, OH-LIGANDS,
ORGANIC ANIONS

Figure 11-16. Schematic representation of the hydrogen ion cycle (Driscoll, 1980).

October 1974, over 70 percent had pH levels below 6.0, 56 percent below 5.5, and 24 percent below 5.0 (Wright and Henriksen, 1978). Of 700 lakes in the Sørlandet Region of southern Norway surveyed in 1974 to 1975 (May-November), 65 percent had pH levels below 5.0 (Wright ad Snekvik, 1978). On the west coast of Sweden, of 321 lakes investigated during 1968-1970, 93 percent had a pH level 5.5 or lower. Fifty-three percent had pH levels between 4.0 and 4.5 (Dickson, 1975). In the LaCloche Mountain Region of Ontario, 47 percent of 150 lakes sampled in 1971 had pH levels less than 5.5, and 22 percent had pH levels below 4.5 (Beamish and Harvey, 1972). In the Adirondacks, 52 percent of the high elevation (> 610 m) lakes had pH values below 5.0 (Schofield, 1976b). In each of these studies, the pH level of an individual lake could be related to, in most cases, the intensity of the acidic deposition and the geologic environment of the watershed. Atmospheric contributions of sea salts are also important in coastal regions.

Several methods have been developed to assess the degree of acidification in a lake and relate it to inputs of hydrogen ion or sulfate. Henriksen (1979) utilized alkalinity-calcium and pH-calcium relationships in lakes to estimate the degree of acidification experienced by a surface water. This technique is based on the premise that when carbonic acid weathering occurs one equivalent of alkalinity (acid neutralizing capacity) is released to the aquatic environment for every equivalent of basic cation (Ca, Mg, K, or Na) dissolved. On the other hand, if mineral acid weathering is occurring, for example as a result of acidic deposition, one equivalent of hydrogen ion is comsumed for every equivalent of cation solubilized. Therefore, for a given basic cation level, there is less aqueous acid neutralizing capacity in lakes in systems experiencing strong acid weathering than in systems experiencing carbonic acid weathering. When comparing alkalinity plots from two watersheds, one experiencing strong acid contributions and the other undergoing largely carbonic acid weathering (assuming both watersheds have similar edaphic environments), the difference in alkalinity between the two plots for a given calcium level (the dominant basic cation) should be indicative of the amount of strong acid the watershed receives and the degree of acidification of the surface water. For waters with pH levels below 5.6, alkalinity is approximately equal to the negative of the hydrogen ion concentration. Therefore, pH level can be substituted for alkalinity, and pH-calcium plots developed (Figure 11-17). Data of this type for Norway indicate that significant acidification of lakes has occurred in areas receiving precipitation with volume-weighted average concentrations of H^+ above 20-25 μeq/liter (pH 4.7-4.6) and sulfate concentrations above 1 mg/liter (20 μeq/liter) (Henriksen, 1979).

Henriksen (1979) also utilized the concentration of excess sulfate in lake water (sulfate in excess of that of marine origin) to estimate acidification. This suggests that bicarbonate anions lost in acidified lakes have been replaced by an equivalent amount of sulfate. Almer et al. (1978) plot pH levels in Swedish lakes as a function of excess sulfur load (excess sulfur in lake water multiplied by the yearly runoff) (Figure 11-18). Based on this relationship, they estimate that the most sensitive lakes in Sweden may resist a load of only about

TABLE 11-4. CHEMICAL COMPOSITION (MEAN ± STANDARD DEVIATION) OF ACID LAKES (pH < 5) IN REGIONS RECEIVING HIGHLY ACIDIC PRECIPITATION (pH <4.5), AND OF SOFT-WATER LAKES IN AREAS NOT SUBJECT TO HIGHLY ACIDIC PRECIPITATION (pH > 4.8)

Region		No. of lakes	Specific conductance††	H (pH)	Na	K	Ca	Mg	μeq/l HCO$_3$	Cl	SO$_4$	NO$_3$	Σ cations	Σ anions	Reference
I LAKES IN ACID AREAS															
Scandinavia															
Southernmost Norway	Measured: Less s.w.*	26	27±10	18±11 (4.76) 18	70±40 9	5±3 4	56±35 50	41±16 25	11±26 11	71±45 0	100±33 97	4±2 4	189 106	186 107	Gjessing et al., 1976
Westcoast Sweden	Measured: Less s.w.*	17	72**	43** (4.37**) 43**	130 <50	70 11	——— 200 ——— -	 -	0 0	440 0	200 155	8 8	673 -	640 -	Hornström et al., 1976
West-central Sweden	Measured: Less s.w.*	6	47±23	22±15 (4.66) 22	165±120 20	15±8 12	75±10 70	80±40 50	- -	170±90 0	200±70 180	19±4 19	360 175	390 200	Grahn et al., 1976
North America															
La Cloche Mtn., Ontario	Measured: Less s.w.*	4	38±8	20±9 (4.7) 20	26±4 9	10±3 10	150±25 150	75±8 65	0 0	22±6 0	290±40 290	- -	280 255	310 290	Beamish, 1976
Sudbury, Ontario	Measured: Less s.w.*	4	120±40	36±15 (4.5) 36	100±30 50	40±10 40	450±180 450	330±120 300	8±2 8	50±20† 0	800±290 800	- -	960 880	850 800	Armstrong, 1971
II LAKES IN UNAFFECTED AREAS															
Scandinavia															
West-central Norway	Measured: Less s.w.*	23	13±3	6±2 (5.2) 6	50±20 9	3±1 3	18±9 16	16±5 7	13±8 13	46±21 0	33±8 30	5±2 5	93 41	97 48	Gjessing et al., 1976
North America															
Experimental Lakes Area, Ontario	Measured: Less s.w.*	40	19	0.2-2 (5.6-6.7) 0.2-2	40 4	10 10	80 80	75 65	60 60	40 0	60 55	<1.5 <1.5	200 160	160 120	Armstrong, 1971

*Less s.w.: Concentrations after subtracting the seawater contribution according to the procedure explained by Wright and Gjessing (1976)
**Data for 117 lakes
†Measured after past liming of the lakes
††μS/cm at 20°C

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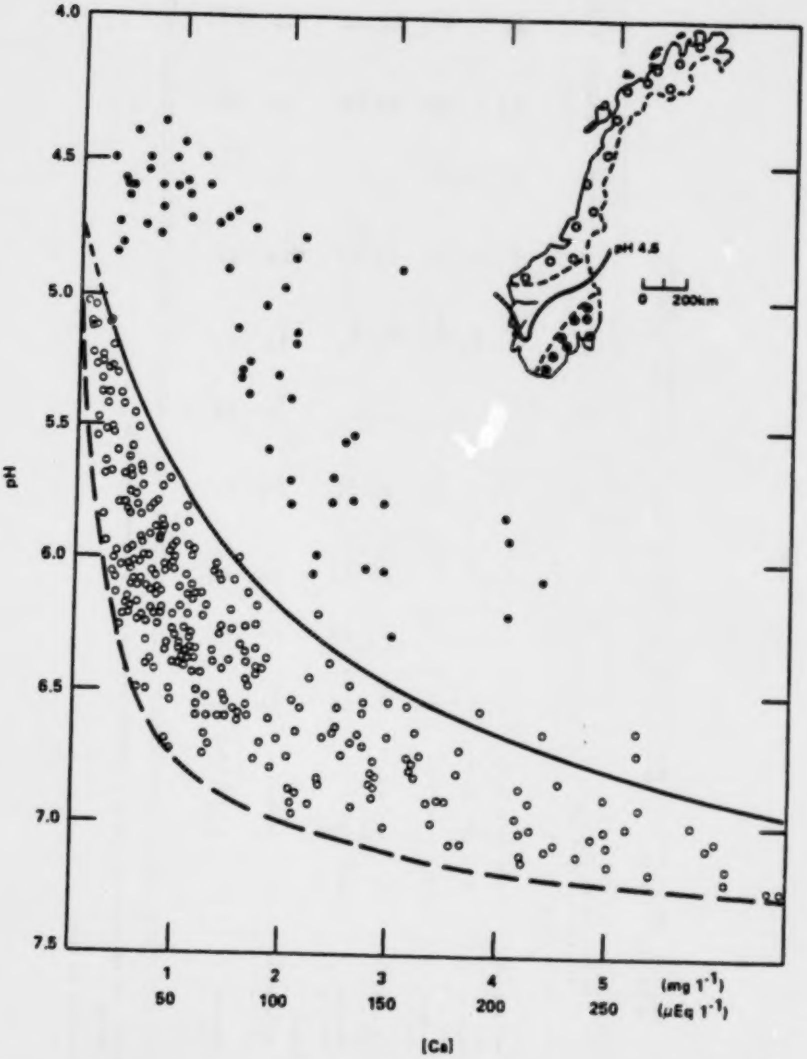

Figure 11-17. pH and calcium concentrations in lakes in northern and northwestern Norway sampled as part of the regional survey of 1975, in lakes in northwestern Norway sampled in 1977 (○) and in lakes in southernmost and southeastern Norway sampled in 1974 (●). Southern Norway receives highly acid precipitation (pH 4.2-4.5) and a large number of lakes have lost their fish populations due to high acidity. Inset shows areas in which these lakes are located. Areas south of isoline receive precipitation more acid than pH 4.6 (Henriksen, 1979).

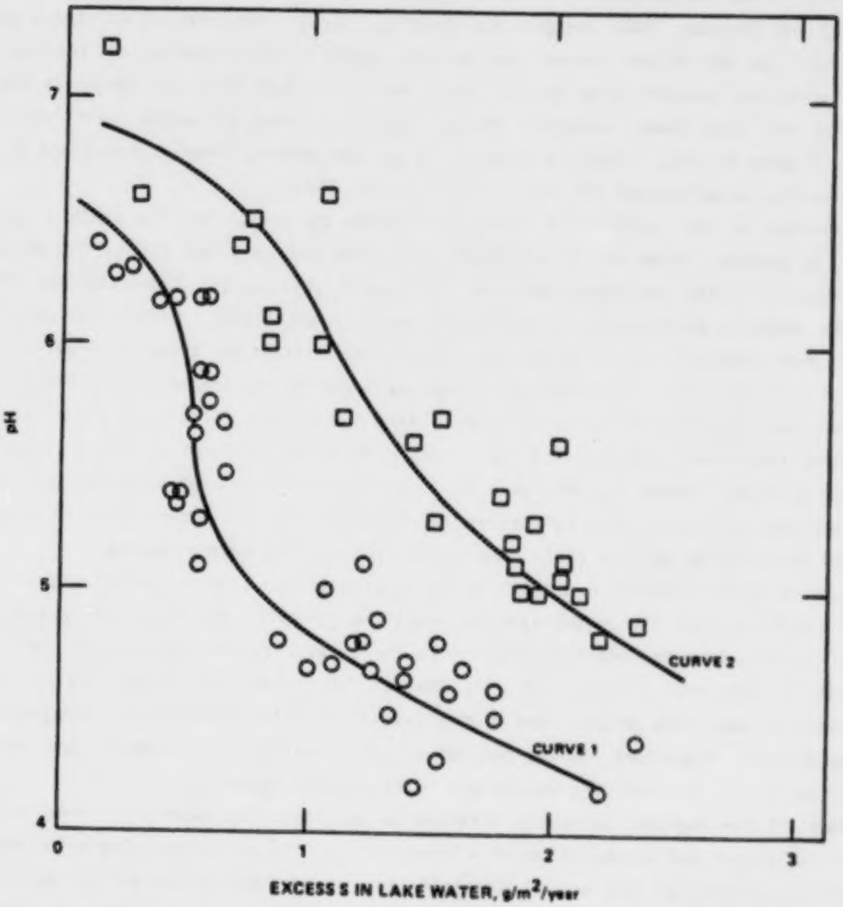

Figure 11-18. The pH value and sulfur loads in lake waters with extremely sensitive surroundings (curve 1) and with slightly less sensitive surroundings (curve 2). (Load = concentration of "excess" sulfur multiplied by the yearly runoff.) (Almer et al., 1978).

0.3 g/m^2 of sulfur in lake water each year. At 1 g/m^2 of sulfur, the pH level of the lake will probably decrease below 5.0.

Elevated metal concentrations (e.g., aluminum, zinc, manganese, and/or iron) in surface waters are often associated with acidification (Beamish, 1976; Hutchinson et al., 1978; Schofield and Trojnar, 1980; Wright and Gjessing, 1976). Mobility of all these metals is increased at low pH values (Stumm and Morgan, 1970). For example, an inverse correlation between aluminum concentration and pH level has been identified for lakes in the Adirondack Region of New York State, southern Norway, the west coast of Sweden, and Scotland (Wright, 1980b). (Figure 11-19). Aluminum appears to be the primary element mobilized by strong acid inputs in precipitation and dry deposition (Cronan, 1978).

Aluminum is the third most abundant element by weight in the earth's crust (Foster, 1971). In general, aluminum is extremely insoluble and retained within the edaphic environment. However, with increased hydrogen ion inputs (via acidic deposition or other sources) into the edaphic environment, aluminum is rapidly mobilized. Cronan and Schofield (1979) suggest that input of strong acids may inhibit the historical trend of aluminum accumulation in the B soil horizon. Consequently, aluminum tends to be transported through the soil profile and into streams and lakes. Evidence from field data (Schofield and Trojnar, 1980) and laboratory experiments (Driscoll et al., 1979; Muniz and Leivestad, 1980) suggest that these elevated aluminum levels may be toxic to fish. Concentration of aluminum may be as or more important than pH levels as a factor leading to declining fish populations in acidified lakes. Aluminum toxicity to aquatic biota other than fish has not been assessed.

Surface water chemistry, particularly in streams and rivers, may be highly variable with time. Since many of the neutralization reactions in soils are kinetically slow, the quality of the leachate from the edaphic system into the aquatic system varies with the retention time of water in the soil (Johnson et al., 1969). The longer the contact period of water with lower soil strata, the greater the neutralization of acid contribution from precipitation and dry deposition. Therefore, during periods of heavy rainfall or snowmelt, and rapid water discharge, pH levels in receiving waters may be relatively depressed.

Many of the regions currently affected by acidification experience freezing temperatures during the winter and accumulation of a snowpack. In the Adirondack Region of New York approximately 55 percent of the annual precipitation occurs during the winter months (Schofield, 1976b). Much of the acid load deposited in winter accumulates in the snowpack, and may be released during a relatively short time period during snowmelt in the spring. In addition, on melting, 50 to 80 percent of the pollutant load (including hydrogen ion and sulfate) may be released in the first 30 percent of the meltwater (Johannessen and Henriksen, 1978). As a result, melting of the snowpack and ice cover can result in a large influx of acidic pollutants into lakes and streams (Figure 11-20) (Gjessing et al., 1976; Hultberg, 1976; Schofield and Trojnar, 1980). The rapid flux of this meltwater through the edaphic environment, and its interaction with only upper soil horizons, limits neutralization of the acid content. As a result, surface waters only moderately acidic during most of the year may experience extreme

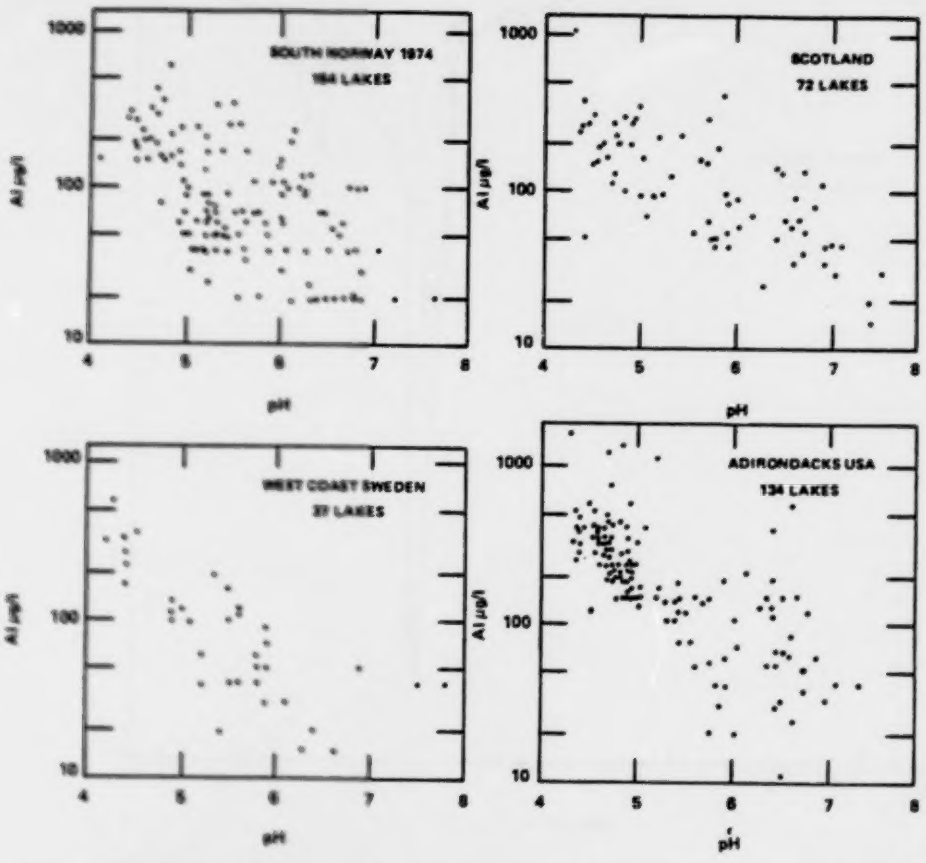

Figure 11-19. Total dissolved Al as a function of pH level in lakes in acidified areas in Europe and North America (Wright et al., 1980b).

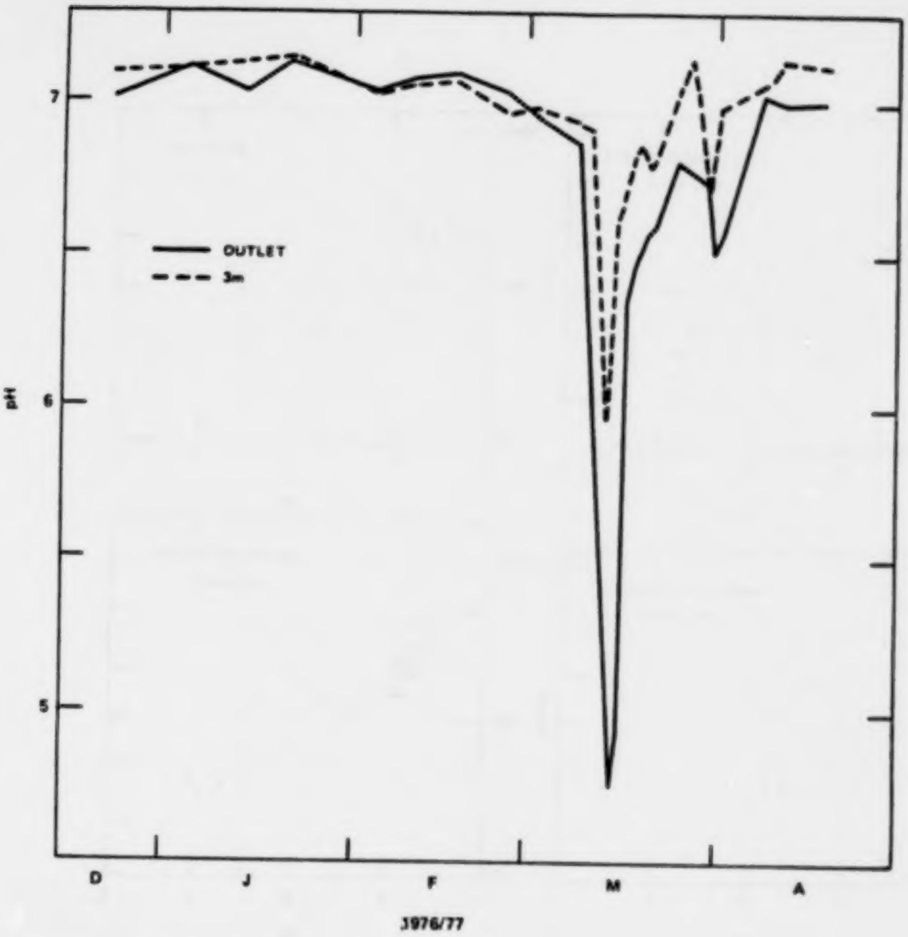

Figure 11-20. pH levels in Little Moose Lake, Adirondack region of New York State, at a depth of 3 meters and at the lake outlet (adapted from Schofield and Trojnar, 1980).

drops in pH level during the spring thaw. Basic cation concentrations (Ca, Mg, Na, K) may also be lower during this time period (Johannessen et al., 1980). Similar but usually less drastic pH drops in surface waters (particularly streams) may occur during extended periods of heavy rainfall (Driscoll, 1980). These short term changes in water chemistry may have significant impacts on aquatic biota.

11.3.1.2 Effects on Decomposition--The processing of dead organic matter (detritus) plays a central role in the energetics of lake and stream ecosystems (Wetzel, 1975). The organic matter may have been generated either internally (autochthonous) via photosynthesis within the aquatic ecosystem or produced outside the lake or stream (allochthonous) and later exported to the aquatic system. Detritus is an important food source for bacteria, fungi, some protozoa, and other animals. These organisms through the utilization of detritus release energy, minerals and other compounds stored in the organic matter back into the environment. Initial processing of coarse particulate detritus is often accomplished by benthic invertebrate fauna. Among other things, the particles are physically broken down into smaller units, increasing their surface area. Biochemical transformations of particulate and dissolved organic matter occur via microbial metabolism and are fundamental to the dynamics of nutrient cycling and energy flux within the aquatic ecosystem.

In general, the growth and reproduction of microorganisms is greatly affected by hydrogen ion concentration (Rheinheimer, 1971). Many bacteria can grow only within the range pH 4-9 and the optimum for most aquatic bacteria is between pH 6.5 and 8.5. There are more acidiphilic fungi than bacteria; consequently in acid waters and sediments the proportion of fungi in the microflora is greater than in waters or sediments with neutral or slightly alkaline pH levels. Most aquatic fungi require free oxygen for growth (Rheinheimer, 1971).

Numerous studies have indicated that acidification of surface waters results in a shift in microbial species and a reduction in microbial activity and decomposition rates. It should be noted, however, that microorganisms in general are highly adaptive. Given sufficient time, a given species may adapt to acid conditions or an acid-tolerant species may invade and colonize acidified surface waters. Therefore, some caution is necessary in interpreting short-term experiments on the effects of acidification on microbial activity and decomposition. On the other hand, increased accumulations of dead organic matter (as a result of decreased decomposition rates) are commonly noted in acidic lakes and streams.

Abnormal accumulations of coarse organic matter have been observed on the bottoms of six Swedish lakes. The pH levels in these lakes in July 1973 were approximately 4.4 to 5.4. Over the last three to four decades, pH levels appear to have decreased 1.4 to 1.7 pH units (Grahn et al., 1974). In both Sweden and Canada, acidified lakes have been treated with alkaline substances to raise pH levels. One result of this treatment has been an acceleration of organic decomposition processes and elimination of excess accumulations of detritus (Andersson et al., 1978; Scheider et al., 1975). Litterbags containing coarse particulate detrital matter have been used to monitor decomposition rates in acidified lakes and streams. In general, the rates of weight loss were reduced in acidic waters when compared with more neutral waters (Leivestad et al., 1978; Traaen, 1980; Peterson, 1980). Traaen (1980) found that after 12 months of incubation dried birch leaves or aspen sticks showed a weight loss of 50-80 percent in waters with pH levels 6 to 7 as compared to only a 30-50 percent weight loss in waters with pH 4 to 5. Petersen (1980) likewise found reduced weight loss of leaf packs incubated in an acidic stream when compared to leaf packs in either a stream not affected by acidification or a stream neutralized with addition of lime. Petersen, however, found no evidence of differences in microbial respiration between the streams. The acidic stream did show a reduction in the invertebrate functional group that specializes in processing large particles (shredders). Gahnstrom et al. (1980) found no significant differences in oxygen consumption by sediments from acidified and non-acidified lakes. Rates of glucose decomposition were also studied in lake sediment-water systems adapted to pH values from 3 to 9. Glucose transformation increased at pH levels above 6. Lime treatment of acidic Lake Högsjön in Sweden also increased rates of glucose processing. However in a humic lake, the maximum rate of glucose transformation occurred at the in situ value pH 5 (Gahnstrom et al., 1980).

Laboratory and field experiments involving decomposition rates have fairly consistently found decreasing microbial activity with increasing acidity. Traaen and Laake (1980) found that litter decomposition at pH level 5.2 was only 50 percent of that at pH 7.0 and at pH 3.5, only 30 percent that at pH 7.0. In addition, increasing acidity (pH 7.0 to 3.5) led to a shift from bacterial to fungal dominance. Incubations of profundal lake sediments at pH 4, 5, and 6 indicated a significant reduction in community respiration with increasing acidity, as well as a possible inhibition of nitrification and a lowering of sediment redox potentials. Bick and Drews (1973) studied the decomposition of peptone in the laboratory. With decreasing pH, total bacterial cell counts and numbers of species of ciliated protozoans decreased, decomposition and nitrification were reduced and oxidation of ammonia ceased below pH 5. At pH 4 and lower, the number of fungi increased.

Disruption of the detrital trophic structure and the resultant interference with nutrient and energy cycling within the aquatic ecosystem may be one of the major consequences of acidification. Investigations into the effects of acidification on decomposition have, apparently, produced somewhat inconsistent results. However, many of these apparent inconsistencies arise only from a lack of complete understanding of the mechanisms relating acidity and rates of decomposition. It is fairly clear that in acidic lakes and streams unusually large accumulations of detritus occur, and these accumulations are related, directly or indirectly, to the low pH level. The processing of organic matter has been reduced. In addition, this accumulation of organic debris plus the development of extensive mats of filamentous algae on lake bottoms (discussed in Section 11.3.1.3) may effectively seal off the mineral sediments from interactions with the overlying water. As a result, regeneration of nutrient supplies to the water column is reduced both by reduced processing and mineralization of dead organic matter and by limiting sediment-water interactions. Primary productivity within the aquatic system may be substantially reduced as a result of this process (Section 11.3.1.3). These ideas have been formulated into the hypothesis of "self-accelerating oligotrophication" by Grahn et al. (1974).

11.3.1.3 Effect on Primary Producers and Primary Productivity--Organisms obtain their food (energy) directly or indirectly from solar energy. Sunlight, carbon dioxide, and water are used by primary producers (phytoplankton, other algae, mosses, and macrophytes) in the process of photosynthesis to form sugars which are used by the plants or stored as starch. The stored energy may be used by the plants or pass through the food chain or web. Energy in any food chain or web passes through several trophic levels. Each link in the food chain is termed a trophic level. The major trophic levels are the primary producers, herbivores, carnivores, and the decomposers. Energy in an ecosystem moves primarily along two main pathways: the grazing food chain (primary producers-herbivores-carnivores) and the detrital food chain (Billings, 1978; Odum, 1971; Smith, 1980). Interactions between these two food chains are, however, extensive. Green plants convert solar energy to organic matter and, as such, are the base for both food chains. The grazing food chain involves primarily living organic matter; the detrital food chain, dead organic matter. Any changes as a result of acidification in the green plants and primary production within the aquatic ecosystem may therefore have a profound effect on all other organisms in the aquatic food web. As noted in Section 11.3.1.2, a portion of the detrital food chain is supported by dead organic matter imported into the aquatic system from external sources.

Extensive surveys of acidic lakes in Norway and Sweden (Almer et al., 1978; Leivestad et al., 1976) have observed changes in species composition and reduced diversity of phytoplankton correlated with decreasing lake pH level (Figure 11-21). Generally at normal pH values of 6 to 8, lakes in the west coast region of Sweden contain 30 to 80 species of phytoplankton per 100-ml sample in mid-August. Lakes with pH below 5 were found to have only about a dozen species. In some very acid lakes (pH<4), only three species were noted. The greatest changes in species composition occurred in the pH interval 5-6. The most striking change was the disappearance of many diatoms and blue-green algae. The families Chlorophyceae (green algae) and Chrysophyceae (golden-brown algae) also had greatly reduced numbers of species in acidic lakes (Figure 11-22). Dinoflagellates constituted the bulk of the phytoplankton biomass in the most acidic lakes (Almer et al., 1978). Similar phenomena were observed in a regional survey of 55 lakes in southern Norway (Leivestad et al., 1976) and in a study of nine lakes in Ontario (Stokes, 1980). Changes in species composition and reduced diversity have also been noted in communities of attached algae (periphyton) (Almer et al., 1978; Leivestad et al., 1976). Mougeotia, a green algae, often proliferates on substrates in acidic streams and lakes.

Shifts in the types and numbers of species present may or may not affect the total levels of primary productivity and algal biomass in acidic lakes. Species favored by acidic conditions may or may not have comparable photosynthetic efficiencies or desirability as a prey item for herbivores. On the other hand, decreased availability of nutrients in acidic water as a result of reduced rates of decomposition (Section 11.3.1.2) may decrease primary productivity

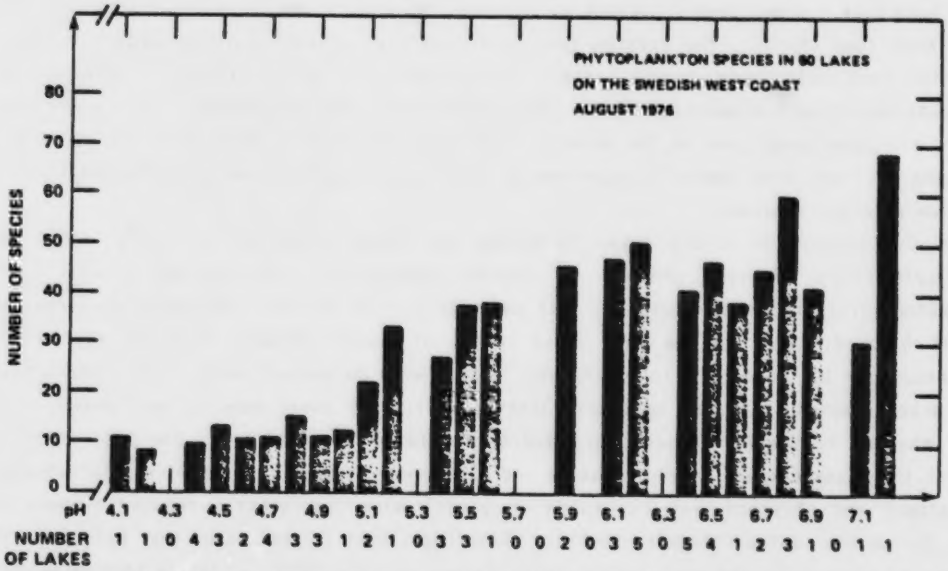

Figure 11-21. Numbers of phytoplankton species in 60 lakes having different pH values on the Swedish West Coast, August 1976 (adapted from Almer et al., 1978).

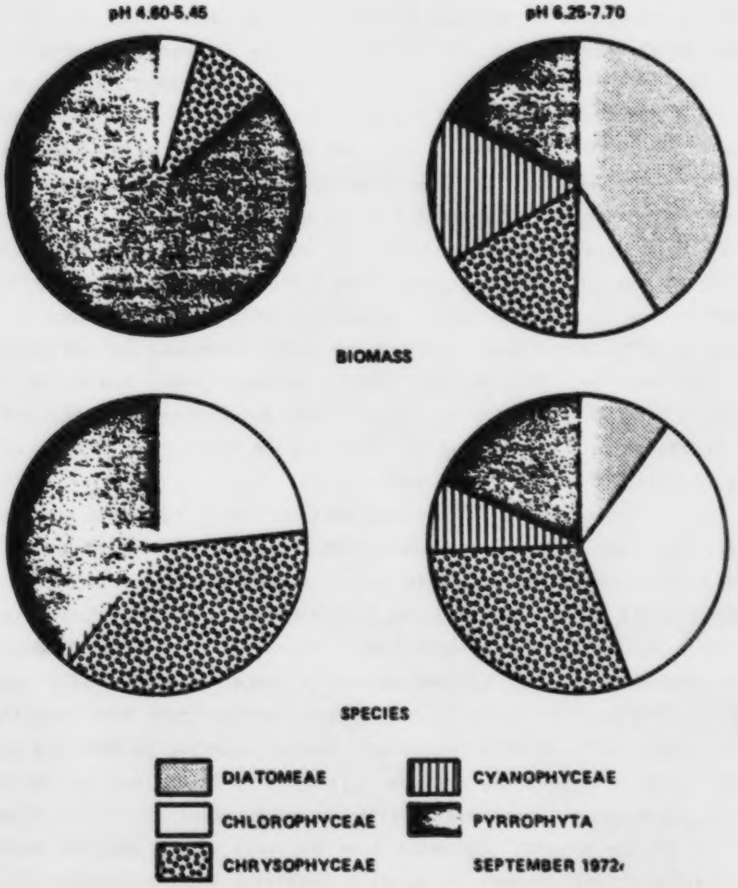

Figure 11-22. Percentage distribution of phytoplankton species and their biomasses. September 1972, west coast of Sweden. Biomass = living weight per unit area (adapted from Almer et al., 1978).

regardless of algal species involved. In field surveys and experiments, relationships between pH level and total algal biomass and/or productivity were not as consistent as the relationship between pH and species diversity.

Kwiatkowski and Roff (1976) identified a significant linear relationship of decreasing chlorophyll a concentrations (indicative of algal biomass) with declining pH level in six lakes near Sudbury, Ontario, with a pH range of 4.05 to 7.15. In addition, primary productivity was reduced in the two most acid lakes (pH 4-4.6). Stokes (1980) also reports a decrease in total phytoplankton biomass with decreasing pH level for nine lakes in the same region of Ontario. Crisman et al. (1980) reported a linear decrease in functional chlorophyll a measurements with declining pH for 11 lakes in northern Florida, pH range 4.5 to 6.9. On the other hand, Almer et al. (1978) note that in 58 nutrient-poor lakes in the Swedish west coast region, the largest mean phytoplankton biomass occurred in the most acid lakes (pH <4.5). Yan and Stokes (1978) concluded that they have no evidence that the phytoplankton biomass in Carlyle Lake, with a summer pH level about 5.1, is below that observed in circumneutral lakes in the same region. In a continuing whole-lake acidification project (Schindler et al., 1980), a lowering of the epilimnion pH level from 6.7-7.0 in 1976 to 5.7-5.9 in 1978 resulted in no significant change in the chlorophyll concentration or primary production. Both in situ and experimental acidification have resulted in large increases in periphyton populations (Hall et al., 1980; Hendrey, 1976; Muller, 1980). Hendrey (1976) and Muller (1980) observed carbon uptake by periphyton incubated in vitro. They found that, although the total rate of photosynthesis increased with decreasing pH level due to the larger biomass at the lower pH, the photosynthesis per unit biomass decreased with pH.

From the above discussion it is obvious that not only is there no clear correlation between pH level and algal biomass or productivity, but the effects of acidification appear inconsistent between systems. Again, these apparent inconsistencies probably reflect a lack of knowledge about exact mechanisms relating acidification and lake metabolism, and also the complexity of these mechanisms and interactions. Changes in the algal community biomass and productivity probably reflect the balance between a number of potentially opposing factors; those that tend to decrease productivity and biomass versus those that tend to increase productivity and/or biomass when acidity increases. Factors working to decrease productivity and biomass with declining pH levels may include: (1) a shift in pH level below that optimum for algal growth, (2) decreased nutrient availability as a result of decreased decomposition rates and a sealing-off of the mineral sediments from the lake water; and (3) decreased nutrient availability as a result of changes in aquatic chemistry with acidification. For example, despite the fact that the optional pH range for growth of Tabellaria flocculosa is between 5.0 to 5.3 (Cholonsky, 1968) or higher (Källqvist et al., 1975), this species dominated experimentally acidified stream communities at pH level 4 in three out of five replicates (Hendrey et al., 1980). As noted in Section 11.3.1.1, aluminum concentrations increase with decreasing pH level in acidified lakes and streams. Aluminum is also a very effective precipitator of phosphorus, particularly in the pH range 5 to 6 (Dickson, 1978; Stumm and Morgan, 1970). In oligotrophic lakes, phosphorus is most commonly the limiting nutrient for primary productivity (Schindler, 1975; Wetzel, 1975). Therefore, chemical interactions between aluminum and phosphorus may result in a decreasing availability of phosphorus with decreasing pH level, and, as a result, decreased primary production.

Factors working to increase productivity and/or biomass with acidification of a lake or stream may include: (1) decreased loss of algal biomass to herbivores; (2) increased lake transparency; and (3) increased nutrient availability resulting from nutrient enrichment of precipitation. Decreased population of invertebrates (as discussed in Section 7.3.1.4), particularly herbivorous invertebrates, may decrease grazing pressure on algae and result in unusual accumulations of biomass. Hendrey (1976) and Hall et al. (1980) include this mechanism as one hypothesis to explain their observation of increased biomass of periphyton at pH level 4 despite a decreased production rate per unit biomass.

Increases in lake transparency over time have been correlated with lake acidification in Sweden (Almer et al., 1978) and the Adirondack Region of New York (Schofield, 1976c). In addition, after the second year of experimental lake acidification (pH 6.7-7.0 to 5.7-5.9) in northwestern Ontario (Schindler et al., 1980), lake transparency increased by 1-2 m. These increases in transparency have not been correlated with decreases in phytoplankton biomass. Two mechanisms have been proposed. Aluminum acts as a very efficient precipitator for humic substances. Dickson (1978) found that humic substances are readily precipitated in the pH range 4.0 to 5.0. Dickson (1978) and Almer et al. (1978) suggest that increases in aluminum levels with lake acidification (Section 11.3.1.1) have resulted in increased precipitation of humics from the water column and therefore increased lake transparency. Almer et al. (1978) provide data for one lake on the west coast of Sweden. The pH level declined from above 6 to about 4.5 between 1940 and 1975. The secchi disc reading increased from about 3m to about 10m over the same period. Organic matter in the water (as estimated by $KMnO_4$ demand) decreased from 24 to 8 mg/liter from 1958 to 1973. Schindler et al. (1980) on the other hand, found no change in levels of dissolved organic carbon with acidification. Instead, changes in hydrolysis of organic matter with declining pH level may affect the light absorbancy characteristics of the molecules. Levels of particulate organic carbon, and changes with pH level, were not reported by Schindler et al. (1980).

Acidification of precipitation (and dry deposition) has been accompanied by increases in levels of sulfate and nitrate. Both of these are nutrients required by plants. However, as noted above, the primary nutrient limiting primary productivity in most oligotrophic lakes is phosphorus. Almer et al. (1978) report that atmospheric deposition rates of phosphorus have also increased in recent years. The world-wide extent of the correlation between acidic deposition and increased atmospheric phosphorus loading, however, is not known. It is expected that changes in atmospheric phosphorus loading would be much more localized than changes in acidic deposition. It is possible that in some areas increased atmospheric loading of phosphorus has occurred in recent years coincidently with increased acidic deposition. Increased phosphorus nutrient loading into lakes may then increase primary production rates.

The effect of acidification on primary productivity and algal biomass of a particular stream or lake system depends upon the balance of the above forces. Differences in the importance of these factors between systems may account for inconsistencies in the response of different aquatic systems to acidic deposition. Acidification does, however, result in a definite change in the nutrient and energy flux of the aquatic system, and this change may eventually limit the total system biomass and productivity.

Acidification of lakes has also been correlated with changes in the macrophyte community. Documentation for these changes comes mainly from lakes in Sweden. Grahn (1976) reported that in five to six lakes studies in the last three to five decades the macrophyte communities dominated by *Lobelia* and *Isoetes* have regressed, whereas communities dominated by *Sphagnum* mosses have expanded. Acidity levels in these lakes apparently have increased approximately 1.3 to 1.7 pH units since the 1930-40's. In acid lakes where conditions are suitable the *Sphagnum* peat moss may cover more than 50 percent of the bottom above the 4-m depth, and may also grow at much lower depths (Almer et al., 1978). The *Sphagnum* invasion may start at lake pH levels just below 6 (Almer et al., 1978). Similar growths of *Sphagnum* occur in Norwegian lakes (National Research Council, 1978). Increases in *Sphagnum* as a benthic macrophyte have been documented from one lake in the Adirondack Region of New York (Hendrey and Vertucci, 1980).

Under acid conditions the *Sphagnum* moss appears to simply outgrow flowering plant aquatic macrophytes. In laboratory tests, the growth and productivity of the rooted macrophyte *Lobelia* was reduced by 75 percent at a pH of 4, compared with the control (pH 4.3-5.5). The period of flowering was delayed by ten days at the low pH (Laake, 1976). At low pH levels (pH<5), essentially all the available inorganic carbon is in the form of carbon dioxide or carbonic acid (Stumm and Morgan, 1970). As a result, conditions may be more favorable for *Sphagnum*, an acidophile that is not able to utilize the carbonate ion.

Besides the shift in macrophyte species, the invasion of *Sphagnum* into acid lakes may have four other impacts on the aquatic ecosystem. *Sphagnum* has a very high ion-exchange capacity, withdrawing basic cations such as Ca^{++} from solution and releasing H^+ (Almer et al., 1978; Anschütz and Gessner, 1954). As a result, the presence of *Sphagnum* may intensify the acidification of the system and decrease the availability of basic cations from other biota. Second, dense growths of *Sphagnum* form a biotype that is an unsuitable substratum for many benthic invertebrates (Grahn, 1976). Growths of *Sphagnum* in acidic lakes are also often associated with felts of white mosses (benthic filamentous algae) and accumulations of non-decomposed organic matter. In combination, these organisms and organic matter may form a very effective seal. Interactions between the water column and the mineral sediments, and the potential for recycling of nutrients from the sediments back into the water body, may be reduced (Grahn, 1976; Grahn et a., 1974). These soft bottoms may also be colonized by other macrophytes. In Sweden, Almer et al. (1978) report that growths of *Juncus*, *Sparaganium*, *Utricularia*, *Nuphar*, and/or *Nymphaea*, in addition to *Sphagnum*, may be extensive in acidic lakes. Thus primary production by macrophytes in lakes with suitable bottoms may be very large. Increased lake transparency may also increase benthic macrophyte and algal primary productivity.

11.3.1.4 Effects on Invertebrates--In regional surveys conducted in southern Norway (Hendrey and Wright, 1976), the west coast of Sweden (Almer et al., 1978), the LaCloche Mountain Region of Canada (Sprules, 1975), and near Sudbury, Ontario (Roff and Kwiatkowski, 1977), numbers of species of zooplankton were strongly correlated with pH level (Figure 11-23). Changes in community structure were most noticeable at pH levels below 5. Certain species (e.g., of the genera Bosmina, Cyclops, Diaptomus, and rotiferans, of the genera Polyarthra, Keratella, and Kellicottia) apparently have a high tolerance of acidic conditions and were commonly found in the pH interval 4.4 to 7.9. Others, such as cladocerans of the Daphnia genus, apparently are more sensitive and were only rarely found at pH <6 (Almer et al., 1978).

Similar studies of the relationship between pH level and biomass or productivity of zooplankton are not available. Proposed mechanisms for interactions between lake acidification and zooplankton populations are therefore largely hypothetical.

The species, population size, and productivity of zooplankton are affected both by changes in the quality and quantity of the food supply and shifts in predator populations. Changes in zooplankton species and production in response to changes in fish populations have been clearly demonstrated (Brooks and Dodson, 1965; Dodson, 1974; Walters and Vincent, 1973). Elimination of fish predators often results in dominance of the zooplankton community by large-bodied species. Absence of invertebrate predators (e.g., large-bodied carnivorous zooplankton) as a result of fish predation or other reasons often results in the prevalence of small-bodied species (Lynch, 1979). Surveys of acidic lake waters often have shown the dominance of small-bodied herbivores in the zooplankton community (Hendrey et al., 1980). Fish also often are absent at these pH levels (Section 11.3.1.5). Different zooplankton species may have different physiological tolerances to depressed pH levels (e.g., Potts and Frye, 1979). Food supplies, feeding habits, and grazing of zooplankton may also be altered with acidification as a consequence of changes in phytoplankton species composition and/or decreases in biomass or productivity of phytoplankton. Zooplankton also rely on bacteria and detrital organic matter for part of their food supply. Thus an inhibition of the microbiota or a reduction in microbial decomposition (Section 11.3.1.2) may also affect zooplankton populations. These alternate mechanisms postulate for changes in community structure and/or production of zooplankton communities probably play an important role in zooplankton responses to acidification.

Synoptic and intensive studies of lakes and streams have also demonstrated that numbers of species of benthic invertebrates are reduced along a gradient of decreasing pH level (Almer et al., 1978; Conroy et al., 1976; Leivestad et al., 1976; Roff and Kwiatkowski, 1977; Sutcliffe and Carrick, 1973). In 1500 freshwater localities in Norway studied from 1953-73, snails were generally present only in lakes with pH levels above 6 (Okland, 1980). Likewise Gammarus Lacustris, a freshwater shrimp and an important element in the diet of fish in Scandinavia, was not found at pH levels below 6.0 (Okland, 1980). Experimental investigations

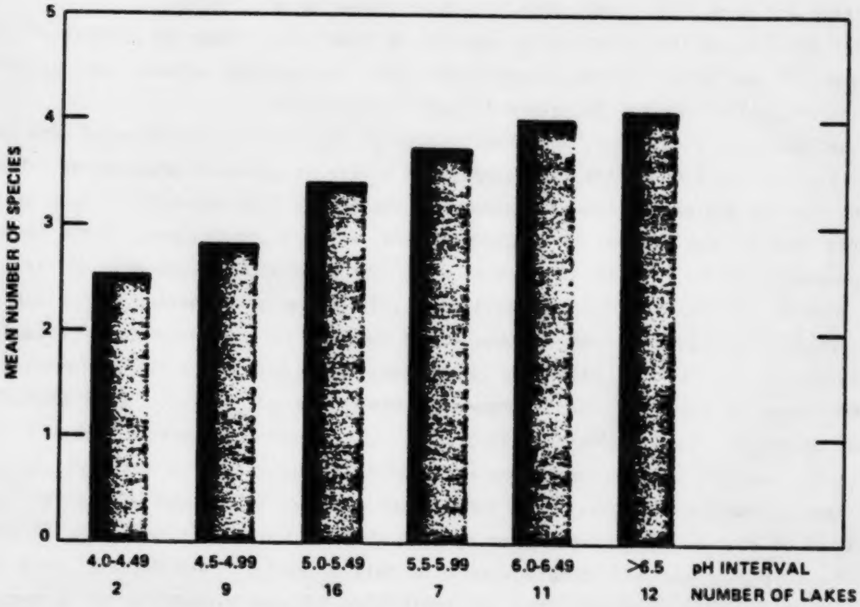

Figure 11-23. The number of species of crustacean zooplankton observed in 57 lakes during a synoptic survey of lakes in southern Norway (Leivestad et al., 1976).

have shown that adults of this species cannot tolerate two days of exposure to pH 5.0 (Leivestad et al., 1976). Eggs were reared at six different pH levels (4.0 to 6.8). At a pH of 4.5 a majority of the embryos died within 24 hours. Thus the short-term acidification which often occurs during the spring melt of snow could eliminate this species from small lakes (Leivestad et al., 1976). Fiance (1977) concluded that ephemeropterans (mayflies) were particularly sensitive to low pH levels and their populations were reduced in headwater streams of the Hubbard Brook watershed in New Hampshire. In laboratory studies, Bell (1971), Bell and Nebecker (1969), and Raddum (1978) measured the tolerance of some stream macroinvertebrates to low pH levels. Tolerance seems to be in the order caddisflies > stoneflies > mayflies (Hendrey et al., 1980).

Leivestad et al. (1976) reported on decreased standing crops (numbers/m^2 and g/m^2) of benthic invertebrates in two lakes with pH levels near 4.5 as compared to five lakes with pH near 6.0. Chironamids were the dominant group in all lakes. No fish were found in the acid lakes. Lack of predation by fish should favor increases in benthic biomass, the opposite of that observed. Hendrey et al. (1980), on the other hand, from data from eight Ontario lakes (pH 4.3 - < 5.7) reported no reduction in abundance of benthos related to pH level.

Air-breathing aquatic insects (e.g., backswimmers, water boatmen, water striders) appear very tolerant of acidic environments. Population densities are often greater in acidic lakes and in the most acid lakes than in circumneutral lakes. Abundance of these large invertebrates may be related to reduced fish predation (Hendrey et al., 1980).

Hall et al. (1980) experimentally acidified a stream to pH 4 and monitored reactions of macroinvertebrate populations. Initially following acidification there was a 13-fold increase in downstream drift of insect larvae. Organisms in the collector and scraper functional groups were affected more than predators. Benthic samples from the acidified zone of Norris Brook contained 75 percent fewer individuals than those for reference areas. There was also a 37 percent reduction in insect emergence; members of the collector group were most affected. Insects seem to be particularly sensitive at emergence (Bell, 1971). Many species of aquatic insects emerge early in the spring through cracks in the ice and snow cover. These early-emerging insects are therefore exposed in many cases to the extremely acidic conditions associated with snowmelt (Hagen and Langeland, 1973).

Low pH also appeared to prevent permanent colonization by a number of invertebrate species, primarily herbivores, in acidified reaches of River Dudden, England (Sutcliffe and Carrick, 1973). Ephemeroptera, trichoptera, Ancylus (Gastropoda) and Gammarus were absent in these reaches.

Damage to invertebrate communities may influence other components of the food chain. Observations that herbivorous invertebrates are especially reduced in acidic streams, as reported in Norris Brook and River Dudden, support the hypothesis (Hall et al., 1980; Hendrey, 1976) that changes in invertebrate populations may be responsible for increased periphytic algal accumulations in acidic streams and benthic regions of acidic lakes (Hendrey et al., 1980). Benthic invertebrates also assist with the essential function of processing dead organic matter. Petersen (1980) noted that decomposition of coarse particulate organic matter in leaf packs was lower in an acidic stream than in two streams with circumneutral pH levels. The invertebrate community also showed a reduction in the invertebrate functional group that specializes in processing large particles (shredders). In unstressed aquatic ecosystems, a continuous emergence of different insect species is available to predators from spring to autumn. In acid-stressed lakes or streams, the variety and numbers of prey may be reduced. Periods may be expected to occur in which the amount of prey available to fish (or other predators) is diminished.

11.3.1.5 Effects on Fish--Acidification of surface waters has had its most obvious, and perhaps the most severe, impact on fish populations. Increasing acidity has resulted not just in changes in species composition or decreases in biomass but in many cases in total elimination of populations of fish from a given lake or stream. Extensive depletion of fish stocks has occurred in large regions of Norway, Sweden, and parts of eastern North America. Both commercial and sport fisheries have been affected in these areas. However, precise assessments of losses--in terms of population extinctions, reductions in yields, or economic and social impacts--either have not been attempted or are still in the process of evaluation. Potential damage to fish populations inhabiting other acid-sensitive aquatic ecosystems in New England, the Appalachians, and parts of southeastern, north central, and northwestern United States have not yet been assessed (National Research Council, 1978).

Declines in fish populations have been related to acidification of surface waters in the Adirondack Region of New York State (Schofield, 1976), southern Norway (Jensen and Snekvik, 1972; Wright and Snekvik, 1978), southwestern Sweden (Almer et al., 1974), the LaCloche Mountain Region in southern Ontario (Beamish and Harvey, 1972), and southwestern Scotland (Wright et al., 1980a). Schofield (1976) estimated that in 1975 fish populations in 75 percent of Adirondack lakes at high elevation (<610 m) had been adversely affected by acidification. Fifty-one percent of the lakes had pH values less than 5, and 90 percent of these lakes were devoid of fish life (Figure 11-24). Comparable data for the period 1929 to 1937 indicated that during that time only about 4 percent of these lakes had pH values below 5 and were devoid of fish (Figure 11-25). Therefore, entire fish communities consisting of brook trout (Salvelinus fontinalis), lake trout (Salvelinus namaycush), white sucker (Catostomus commersoni), brown bullhead (Icaturus nebulosus) and several cyprinid species were apparently eliminated over a period of 40 years. This decrease in fish populations was associated with a decline in lake pH level. A survey of more than 2000 lakes in southern Norway, begun in 1971, found that about one third of these lakes had lost their fish population (primarily brown trout, Salmo trutta L.) since the 1940's (Wright and Snekvik, 1978). Fish population status was inversely related to lake pH level (Leivestad et al., 1976). Declines in salmon populations in southern Norwegian rivers were reported as early as the 1920's. Catch of Atlantic

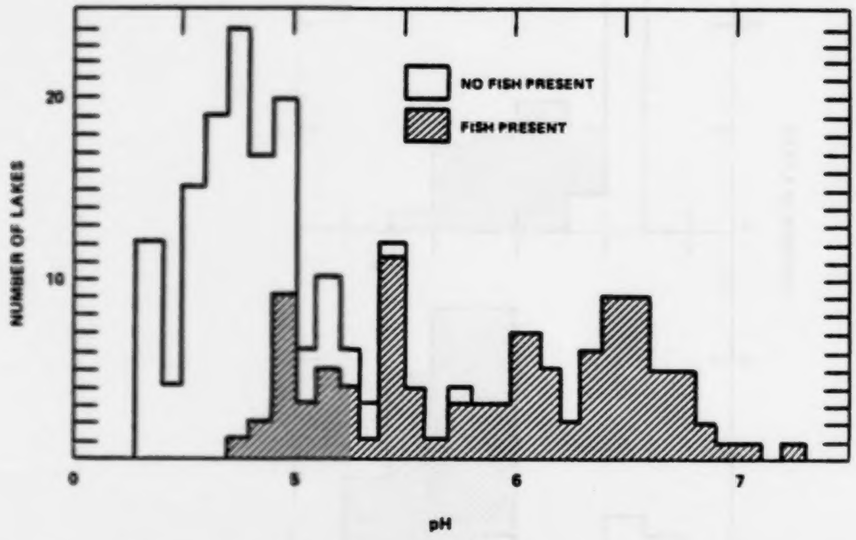

Figure 11-24. Frequency distribution of pH and fish population status in Adirondack Mountain lakes greater than 610 meters elevation. Fish population status determined by survey gill netting during the summer of 1975 (Schofield, 1976b).

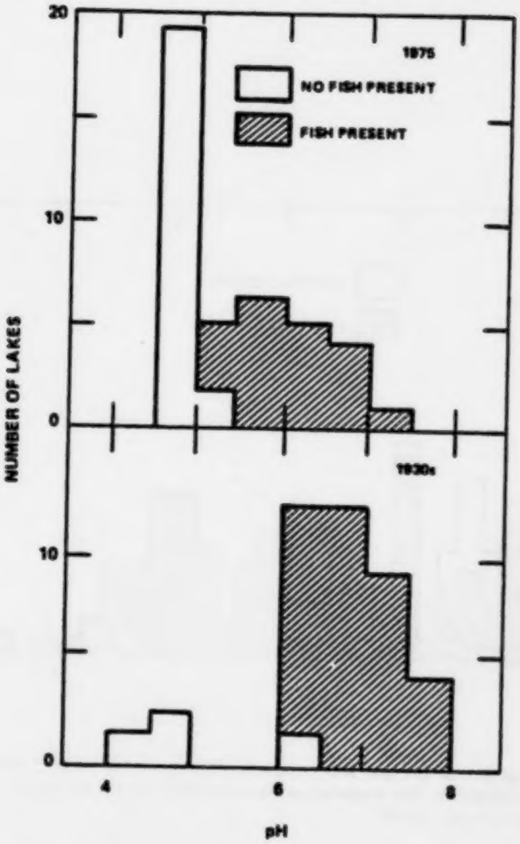

Figure 11-25. Frequency distribution of pH and fish population status in 40 Adirondack lakes greater than 610 meters elevation, surveyed during the period 1929-1937 and again in 1975 (Schofield, 1976b).

salmon (Salmo salar, L.) in nine acidified southern Norwegian rivers is now virtually zero (Figure 11-26). In northern and western rivers not affected by acidification, no distinct downward trend in catch has occurred (Jensen and Snekvik, 1972; Leivestad et al., 1976; Wright et al., 1976). Similar changes have been observed in Sweden (Almer et al., 1974) where it is estimated that 10,000 lakes have been acidified to a pH less than 6.0 and 5,000 below a pH of 5.0 (Dickson, 1975). Populations of lake trout, lake herring (Coregonus artedii), white suckers, and other species disappeared rapidly during the 1960's from a group of remote lakes in the LaCloche Mountain Region of Ontario (Beamish et al., 1975).

It is difficult to determine at what pH level fish species disappear from lakes. Disappearance of the fish is usually not due to massive fish kills, but is the result of a gradual depletion of the population following reproductive failures (Leivestad et al., 1976). Field surveys in Scandinavia and eastern North America (Almer et al., 1974; Schofield, 1976a, 1976b, 1976c; Wright and Snekvik, 1978) suggest that many species do not occur in lakes with pH values below 5.0.

However, large spatial and temporal functions in pH, and the possibility for "refuge areas" from acidic conditions during critical periods make it extremely difficult to generalize about effects of acidification on fish populations based on grab samples or annual mean pH levels. The pH levels identified in the literature as critical for reproduction of a species or correlated with the absence of a species in lake surveys are summarized in Table 11-5. Values range from pH 4.4 to over 6.0, and are highly species dependent.

Recent field and laboratory studies (Baker and Schofield, 1980; Dickson, 1978; Driscoll et al., 1979; Muniz and Leivestad, 1980; Schofield and Trojnar, 1980) have indicated that aluminum levels in acidic surface waters (Section 11.3.1.1, Figure 11-19) may be highly toxic to fish (and perhaps other biota). Schofield and Trojnar (1980) analyzed survival of brook trout stocked into 53 Adirondack lakes as a function of 12 water quality parameters. Levels of pH, calcium, magnesium, and aluminum were significantly different between the two groups of lakes, with and without trout survival. However, after accounting for the effects of aluminum concentrations on differences between the two groups of lakes, differences in calcium, magnesium, and pH levels were no longer significant. Aluminum, therefore, appears to be the primary chemical factor controlling survival of trout in these lakes. Likewise, in laboratory experiments with natural Adirondack waters and synthetic acidified aluminum solutions, levels of aluminum, and not the pH level per se, determined survival and growth of fry of brook trout and white suckers (Baker and Schofield, 1980). In addition, speciation of aluminum had a substantial effect on aluminum toxicity. Complexation of aluminum with organic chelates eliminated aluminum toxicity to fry (Baker and Schofield, 1980; Driscoll et al., 1979). As a result, waters high in organic carbon, e.g., acidic bog lakes, may be less toxic to fish than surface waters at similar pH levels but with lower levels of dissolved organic carbon.

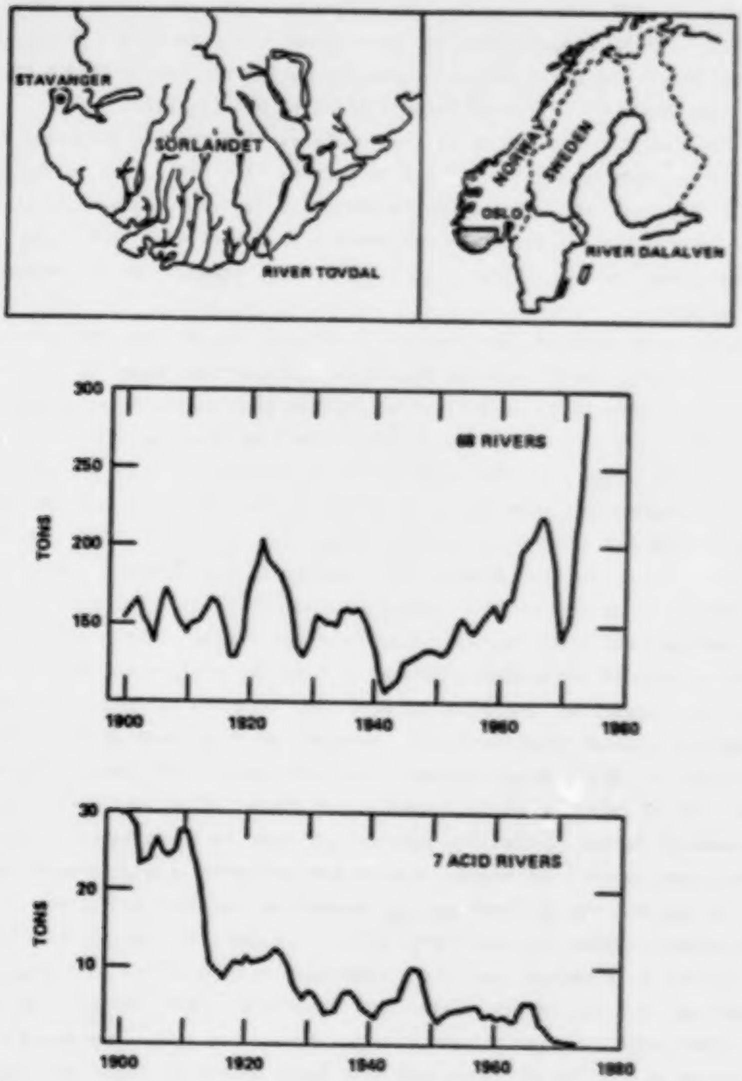

Figure 11-26. Norwegian salmon fishery statistics for 68 unacidified and 7 acidified rivers (adapted from Almer et al., 1978).

TABLE 11-5. pH LEVELS IDENTIFIED IN FIELD SURVEYS AS
CRITICAL TO LONG-TERM SURVIVAL OF FISH POPULATIONS

Family	Species	Critical pH	Reference
Salmonidae	Brook trout (*Salvelinus fontinalis*)	5.0	Schofield, 1976c
	Lake trout (*Salvelinus namaycush*)	5.1 5.2-5.5	Schofield, 1976c Beamish, 1976
	Brown trout (*Salmo trutta*)	5.0	Almer et al., 1978
	Arctic char (*Salvelinus alpinus*)	5.2	Almer et al., 1978
Percidae	Perch (*Perca fluviatilis*)	4.4-4.9	Almer et al., 1978
	Yellow perch (*Perca flavescens*)	4.5-4.7	Beamish, 1976
	Walleye (*Stigostedion vitreum*)	5.5-6.0+	Beamish, 1976
Catostomidae	White sucker (*Catostomus commersoni*)	4.7-5.2 5.1	Beamish, 1976 Schofield, 1976c
Ictaluridae	Brown bullhead (*Icaturus nebulosus*)	4.7-5.2 5.0	Beamish, 1976 Schofield, 1976c
Cyprinidae	Minnow (*Phoxinus phoxinus*)	5.5	Almer et al., 1978
	Roach (*Rutilus rutilus*)	5.5	Almer et al., 1978
	Lake chub (*Couesius plumbeus*)	4.5-4.7	Beamish, 1976
	Creekchub (*Semotilus atromaculatus*)	5.0	Schofield, 1976c
	Commonshiner (*Notropis cornutas*)	5.5	Schofield, 1976c
	Goldenshiner (*Notemigonus crysoleucas*)	4.9	Schofield, 1976c
Centrarchidae	Smallmouth bass (*Micropterus dolomieui*)	5.5-6.0+	Beamish, 1976
	Rock bass (*Ambloplites rupestris*)	4.7-5.2	Beamish, 1976
Esocidae	Pike (*Esox lucius*)	4.4-4.9	Almer et al., 1978

Inorganic aluminum levels, and not low pH levels, may therefore be a primary factor leading to declining fish populations in acidified lakes and streams. However, many laboratory or *in situ* field experiments have been conducted on the effects of pH on fish without taking into account aluminum or other metal concentrations in naturally acidic waters. As a result, many of the conclusions based on these experiments regarding pH levels critical for fish survival are suspect. Therefore these experiments will not be reviewed here.

Sensitivity of fish and other biota to low pH levels has also been shown to depend on aqueous calcium levels (Bua and Snekvik, 1972; Trojnar, 1977; Wright and Snekvik, 1978). In southern Norway, the mean calcium level in lakes studied was approximately 1.1 mg/liter, as compared to about 3 mg/liter in the LaCloche Mountain Region (Table 11-4) or 2.1 mg/liter in the Adirondack Region (Schofield, 1976b). In Norwegian lakes, Wright and Snekvik (1978) identified pH and calcium levels as the two most important chemical parameters related to fish status.

Decreased recruitment of young fish has been cited as the primary factor leading to the gradual extinction of fish populations (Leivestad et al., 1976; Rosseland et al., 1980; Wright and Snekvik, 1978). Field observations (Almer et al., 1974; Beamish, 1974; Jensen and Snekvik, 1972; Schofield, 1976) indicate changes in population structure over time with acidification. Declining fish populations consist primarily of older and larger fish with a decrease in total population density. Recruitment failure may result from inhibition of adult fish spawning and/or increased mortality of eggs and larvae. Effects on spawning and decreased egg deposition may be associated with disrupted spawning behavior and/or effects of acidification on reproductive physiology in maturing adults (Lockhart and Lutz, 1976). Field observations by Beamish et al., (1975) related reproductive failure in white suckers to an inability of females to release their eggs. On the other hand, Amundsen and Lunder (1974) observed total mortality of naturally spawned trout eggs in an acid brook a few weeks after spawning. A summary of Norwegian studies (Leivestad et al., 1976) concluded that egg and fry mortality is the main cause of fish reproduction failure. Spawning periods and occurrence of early life history stages for many fish species coincide with periods of extreme acidity, particularly during and immediately after snowmelt in the spring.

In some lakes, fish population decreases are associated with a lack of older fish (Rosseland et al., 1980). In Lake Tveitvatn on the Tovdal River in southern Norway, brown trout mortality apparently occurs primarily after the first spawning. Since 1976, no fish past spawning age have been found and population density has decreased steadily (Rosseland et al., 1980). Fish kills of adult salmon in rivers in southern Norway have been recorded as early as 1911 (Leivestad et al., 1976).

When evaluating the potential effects of acidification on fish, or other biotic, populations, it is very important to keep in mind the highly diversified nature of aquatic systems spatially, seasonally, and year-to-year. As a result of this diversity, it is necessary to evaluate each system independently in assessing the reaction of the population to acidification. Survival of a fish population may depend more on the availability of refuge areas from acid conditions during spring melt or of one tributary predominantly fed by baseflow and supplying an adequate area for spawning than on mean annual pH, calcium, or inorganic aluminum levels.

11.3.1.6 Effects on Vertebrates Other Than Fish--Certain species of amphibians may be the vertebrate animals, other than fish, most immediately and directly affected by acidic deposition.[194] Their vulnerability is due to their reproductive habits. In temperate regions, most species of frogs and toads, and approximately half of the terrestrial salamanders, lay eggs in ponds. Many of these species breed in temporary pools formed each year by accumulation of rain and melted snow. Approximately 50 percent of the species of toads and frogs in the

United States regularly breed in ephemeral pools; about one-third of the salamander species that have aquatic eggs and larvae and terrestrial adults breed in temporary pools. Most of these pools are small and collect drainage from a limited area. As a result, the acidity of the eater in these pools is strongly influenced by the pH of the precipitation that fills them. Ephemeral pools are usually more acidic than adjacent permanent bodies of water. Pough and Wilson (1976) report that in 1975, in the vicinity of Ithaca, N.Y., the average pH of 12 temporary ponds was 4.5 (range 3.5 to 7.0), while the average pH of six permanent ponds was 6.1 (range 5.5 to 7.0). Amphibian eggs and larvae in temporary pools are exposed to these acidic conditions.

Pough and Wilson (1976) and Pough (1976) studied the effect of pH level on embryonic development of two common species of salamanders: the spotted salamander (Ambystoma maculatum) and the Jefferson salamander (A. jeffersonianum). In laboratory experiments, embryos of the spotted salamander tolerated pH levels from 6 to 10 but had greatest hatching success at pH 7 to 9. The Jefferson salamander tolerated pH levels 4 to 8 and was most successful at 5 to 6. Mortality of embryos rose abruptly beyond the tolerance limits. In a four-year study of a large breeding pond (pH 5.0-6.5) 938 adult spotted salamanders produced 486 metamorphosed juveniles (0.52 juveniles/adult), while 686 adult Jefferson salamanders produced 2157 juvenile (3.14 juveniles/adult). Based on these findings, Pough and Wilson (1976) predict that continued acidic deposition may result in substantial shift in salamander and other amphibian populations.

Gosner and Black (1957) report that only acid-tolerant species of amphibians can breed in the acid (pH 3.6 to 5.2) sphagnoceous bogs in the New Jersey Pine Barrens.

Frog populations in Tranevatten, a lake near Gothenberg, Sweden, acidified by acidic precipitation, have also been investigated (Hogström, 1977; Hendrey, 1978). The lake has pH levels ranging from 4.0 to 4.5. All fish have disappeared, and frogs belonging to the species Rana temporaria and Bufo bufo are being elimated. At the time of the study (1977) only adult frogs eight to ten years old were found. Many egg masses of Rana temporaria were observed in 1974, but few were found in 1977, and the few larvae (tadpoles) observed at that time died.

Frogs and salamanders are important predators on invertebrates, such as mosquitoes and other pest species, in pools, puddles, and lakes. They also are themselves important prey for higher tropic levels in an ecosystem. In many habitats salamanders are the most abundant vertebrates. In a New Hampshire forest, for example, salamanders were found to exceed birds and mammals in both numbers and biomass (Hanken et al., 1980).

The elimination of fish and vegetation from lakes by acidification may have an indirect effect on a variety of vertebrates: species of fish-eating birds (e.g., the bald eagle, loon, and osprey), fish-eating mammals (e.g., mink and otter), and dabbling ducks which feed on aquatic vegetation. In fact, any animal that depends on aquatic organisms (plant or animal) for a portion of its food may be affected.

Increasing acidity in freshwater habitats results in shifts in species, populations, and communities. Virtually all trophic levels are affected.

11.3.2 Terrestrial Ecosystems

Determining the effects of acidic precipitation on terrestrial ecosystems is not an easy task. In aquatic ecosystems it has been possible to measure changes in pH that occur in acidified waters and then observe the response of organisms living in aquatic ecosystems to the shifts in pH. In the case of terrestrial ecosystems the situation is more complicated since no component of terrestrial ecosystems appears to be as sensitive to acidic precipitation as organisms living in poorly buffered aquatic ecosystems. Nonetheless, soils and vegetation may be affected, directly or indirectly, by acidic precipitation, albeit in complex ways.

11.3.2.1 Effects on Soils--Acidity is a critical factor in the behavior of natural or agricultural soils. Soil acidity influences the availability of plant nutrients and various microbiological processes which are necessary for the functioning of terrestrial ecosystems, therefore, there is concern that acidic precipitation over time could have an acidifying effect on soils through the addition of hydrogen ions. As water containing hydrogen cations (usually from weak acids) moves through the soil, some of the hydrogen ions replace adsorbed exchangeable cations, such as Ca^{++}, Mg^{++}, K^+, and Na^+ (see Figure 11-27). The removed cations are then carried deep into the soil profile or into the ground water. In native soils hydrogen ions are derived from the following sources: (Wiklander, 1979)

1. nutrient uptake by plants--the roots adsorb cation nutrients and desorb H^+;
2. CO_2 produced by plant roots and micro-organisms;
3. oxidation of NH_4^+ and S, FeS_2, and H_2S to HNO_3 and H_2SO_4;
4. very acid litter in coniferous forests, the main acidifying source for the A and B horizons;
5. atmospheric deposition of H_2SO_4 and some HNO_3, NO_x, HCl and NH_4^+ (after nitrification to HNO_3).

In addition to the acidifying factors listed above, the use of ammonium fertilizers on cultivated lands increases the hydrogen cations in the water solution. Ammonium fertilizers are oxidized by bacteria to form nitrate (NO_3^-) and hydrogen ions (H^+) (Donahue et al., 1977). Increased leaching causes soils to become lower in basic Ca^{++}, Mg^{++}, Na^+, and K^+ cations (Donahue et al., 1977). Sensitivity to leaching is according to the following sequence: $Na^+ >> K^+ > Mg^{2+} > Ca^{2+}$ (Wiklander, 1979).

Norton (1977) cited the potential effects of acidic deposition on soils that are listed in Table 11-6. Of those listed, only the increased mobility of cations and their accelerated loss has been observed in field experiments. Overrein (1972) observed an increase in calcium leaching under simulated acid rain conditions and increased loss by leaching of Ca^{++}, Mg^{++},

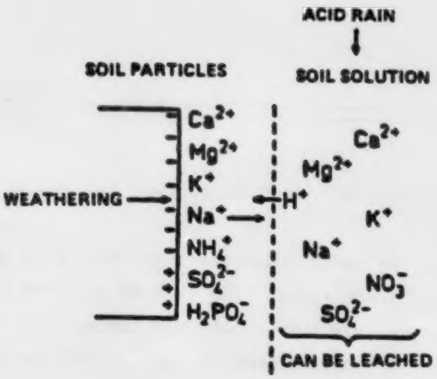
ACID RAIN
SOIL PARTICLES
SOIL SOLUTION
Ca^{2+}
Mg^{2+}
K^+
WEATHERING
Na^+
NH_4^+
SO_4^{2-}
$H_2PO_4^-$
H^+
Ca^{2+}
Mg^{2+}
K^+
Na^+
NO_3^-
SO_4^{2-}
CAN BE LEACHED

Figure 11-27. Showing the exchangeable ions of a soil with pH 7, the soil solution composition, and the replacement of Na^+ by H^+ from acid rain (Wiklander, 1979).

TABLE 11-6. POTENTIAL EFFECTS OF ACID PRECIPITATION ON SOILS

Effect	Comment
Increased mobility of most elements	Mobility changes are essentially in the order: monovalent, divalent, trivalent cations.
Increased loss of existing clay minerals	Under certain circumstances may be compensated for by production of clay minerals which do not have essential (stoichiometric) alkalies or alkali earths.
A change in cation exchange capacity	Depending on conditions, this may be an increase or a decrease.
A general proportionate increase in the removal of all cations from the soil	In initially impoverished or unbuffered soil, the removal may be significant on a time scale of 10 to 100 years.
An increased flux in nutrients through the ecosystem below the root zone	

Source: Norton (1977).

and Al^{+3} were observed by Cronan (1980) when he treated New Hampshire soils with simulated acid rain at a pH 4.4.

Wiklander (1979) notes that in humid areas leaching leads to a gradual decrease of plant nutrients in available and mobilizable forms. The rate of nutrient decrease is determined by the buffering capacity of the soil and the amount and composition of precipitation (pH and salt content). Leaching sooner or later leads to soil acidification unless the buffering capacity of the soil is strong and/or the salt concentration of precipitation is high. Soil acidification influences the amount of exchangeable nutrients and is also likely to affect various biological processes in the soil.

Acidic precipitation increases the amounts of SO_4^{2-} and NO_3^- entering the soils. Nitrate is easily leached from soil; however, because it is usually deficient in the soil for both plants and soil microorganisms, it is rapidly taken up and retained within the soil-plant system (Abrahamsen et al., 1976; Abrahamsen and Dollard, 1979; Gjessing et al., 1976). The fate of sulfate is determined by its mobility. Retention of sulfate in soils appears to depend on the amount of hydrous oxides of iron and aluminum present. The amounts of these compounds present varies with the soil type. Insignificant amounts of the hydrated oxides of iron (Fe) and aluminum (Al) are found in organic soils; therefore, sulfate retention is low (Abrahamsen and Dollard, 1979). The presence of hydrated oxides of iron and aluminum, however, is only one of the factors associated with the capability of a soil to retain sulfur. The capacity of soils to adsorb and retain anions increases as the pH decreases and with the salt concentration. Polyvalent anions of soluble salts added experimentally to soils increases adsorption and decreases leaching of salt cations. The effectiveness of the anions studied in preventing leaching was in the following order: $Cl^- \sim NO_3^- < SO_4^{2} < H_2PO_4^-$ (Wiklander, 1980). Additions of sulfuric acid to a soil will have no effect on cation leaching unless the sulfate is mobile, as cations cannot leach without associated anions (Johnson et al., 1980; Johnson, 1980; Johnson and Cole, 1980).

Leaching of soil nutrients is efficiently inhibited by vegetation growing on it. Plant roots take up the nutrients frequently in larger amounts than required by the plants. Large amounts of these nutrients will later be deposited on the soil surface as litter or as leachate from the vegetation canopy (Abrahamsen and Dollard, 1979).

In lysimeter experiments in Norway, plots with vegetation cover were used. One plot had a dense layer of the grass, *Deschampsia flexuosa* (L.) Trin. and the other a less dense cover. The soil retained 50 percent of the SO_4^{2-} added to it. The greatest amount was retained in the lysimeters covered with grass; the relative retention increased with increasing additions of sulfate (Abrahamsen and Dollard, 1979). Leaching of cations from the soil was reduced by the retention of the SO_4^{2-}; however, leaching of Ca^{2+} and Mg^{2+} increased significantly as the acidity of the simulated rain increased. In the most acid treatment leaching of Al was highly significant. The behavior of K^+, NO_3^-, and NH_4^+ was different in the two lysimeter series. These ions were retained in the grass-covered lysimeters whereas there was a net leaching of K^+ and NO_3^- in the other series. Statistically significant effects were obtained only when the pH of the simulated rain was 3.0 or lower (Abrahamsen and Dollard, 1979).[2]

The Scandinavian lysimeter experiments appear to demonstrate that the relative rate of adsorption of sulphate increases as the amounts applied are increased. In the control lysimeters the output/input ratio was approximately one. These results are in agreement with results of watershed studies which frequently appear to demonstrate that, on an annual basis, sulfate outflow is equal to or greater than the amounts being added (Abrahamsen and Dollard, 1979; Gjessing et al., 1976). Increased outflow may be attributed to dry deposition and the weathering of sulfur-bearing rocks. The increased deposition of sulfate via acidic precipitation appears to have increased the leaching of sulfate from the soil. Together with the retention of hydrogen ions in the soil this results in an increased leaching of the nutrient cations K^+, Ca^{2+}, Mg^{2+}, Mn (Abrahamsen and Dollard, 1979). Shriner and Henderson, (1978) however, in their study of sulfur distribution and cycling in the Walker Branch Watershed in eastern Tennessee noted the additions of sulfate sulfur by precipitation were greater than the amount lost in stream flow. Analysis of the biomass and soil concentrations of sulfur indicated that sulfur was being retained in the mineral soil horizon. It is suggested that leaching from organic soil horizons may be the mechanism by which sulfur is transferred to the mineral horizon. Indirect evidence suggests that vegetation scavenging of atmospheric sulfate plays an important role by adding to the amounts of sulfur entering the forest system over wet and dry deposition.

Studies of the nutrient cycling of sulfur in a number of forest ecosystems indicate that some ecosystems accumulate (Heinricks and Mayer, 1977; Johnson et al., 1980; Shriner and Henderson, 1978) while other ecosystems maintain a balance between the additions and losses of sulfur or show a net loss (Cole and Johnson, 1977). Sulfur accumulation appears to be associated with sulfate adsorption in subsoil horizons. Sulfate adsorption is strongly dependent on pH. Little adsorption occurs above pH 6-7 (Harward and Reisenaur, 1966). The amount of sulfate in a soil is a function of a soil's adsorption properties and the amount of sulfate that has been added to the soil, integrated over time. Soil properties may favor the adsorption of sulfate; however, the net annual accumulation of sulfate at any specific time will be influenced by the degree of soil saturation (Johnson et al., 1980).

The effects of acidic precipitation on soils potentially could be long-lasting. Oden (1971) has estimated that rainfall at pH 4.0 would be the cation equivalent of 30 kg Ca^{+2}/ha, which represents a considerable potential loss of cations essential for plant growth as well as base saturation. McFee et al. (1976) calculated that 1000 cm of rainfall at pH 4.0 could reduce the base saturation of the upper 6 cm of a midwestern United States forest soil by 15 percent and lower the pH of the A-1 horizon (the surface layer in most agricultural soils) by 0.5 units if no countering forces are operating in the soil. They note, however, that many counteracting forces could reduce the final effect of acidic precipitation, including the release of new cations to exchange sites by weathering and nutrient recycling by vegetation.

Lowered soil pH also influences the availability and toxicity of metals to plants. In general, potentially toxic metals become more available as pH decreases. Ulrich (1975) reported that aluminum released by acidified soils could be phytotoxic if acid rain continued for a long period. The degree of ion leaching increased with decreases in pH, but the amount of cations leached was far less than the amount of acid added (Malmer, 1976). Baker et al. (1976) found that sulfur dioxide in precipitation increased the extractable acidity and aluminum, and decreased the exchangeable bases, especially calcium and magnesium. Although dilute sulfuric acid in sandy podsolic soils caused a significantly decreased pH of the leached material, the amount of acid applied (not more than twice the yearly airborne supply over southern Scandinavia) did not acidify soil as much as did nitrate fertilizer (Tamm et al., 1977). Highly acidic rainfall, frequently with a pH less than 3.0, in combination with heavy metal particulate fallout from smelters, has caused soils to become toxic to seedling survival and establishment according to observations by Hutchinson and Whitby (1976). Very low soil pH's are associated with mobility of toxic aluminum compounds in the soils. High acidity, high sulfur, and heavy metals in the rainfall have caused fundamental changes in the structure of soil organic matter. The sulfate and heavy metals were borne by air from the smelters in the Sudbury area of Ontario and brought to earth by dry and wet deposition. Among the metals deposited in rainfall and dustfall were nickel, copper, cobalt, iron, zinc, and lead. Most of these metals are retained in the upper layers of soil, except in very acid or sandy soils.

The accumulation of metals is mainly an exchange phenomenon. Organic components of litter, humus, and soil may bind heavy metals as stable complexes (Tyler, 1972). The heavy metals when bound may interfere with litter decay and nutrient cycling, and in this manner interfere with ecosystem functioning (Tyler, 1972). Acidic precipitation, by altering the equilibria of the metal complexes through mobilization, may have a negative effect upon the residence time of the heavy metals in soil and litter (Tyler, 1972, 1977).

Biological processes in the soil necessary for plant growth can be affected by soil acidification. Nitrogen fixation, decomposition of organic material, and mineralization, especially of nitrogen, phosphorus and sulfur, might be affected (Abrahamsen and Dollard, 1979; Alexander, 1980; Malmer, 1976; Tamm et al., 1977). Nearly all of the nitrogen, most of the phosphorus and sulfur as well as other nutrient elements in the soil are bound in organic combination. In this form, the elements are largely or entirely unavailable for utilization by higher plants (Alexander, 1980). It is principally through the activity of heterotrophic microorganisms that nitrogen, phosphorus, and sulfur are made available to the autotrophic higher plants. Thus, the microbial processes that lead to the conversion of the organic forms of these elements to the inorganic state are crucial for maintaining plant life in natural or agricultural ecosystems. The key role of these degradative processes is the fact that nitrogen is limiting for food production in much of the world and governs primary productivity in many terrestrial habitats (Alexander, 1980).

Many, and probably most, microbial transformations in soil may be brought about by several species. Therefore, the reduction or elimination of one population is not necessarily detrimental since a second population, not affected by the stress, may fill the partially or totally vacated niche. For example, the conversion of organic nitrogen compounds to inorganic forms is characteristically catalyzed by a number of species, often quite dissimilar, and a physical or chemical perturbation affecting one of the species may not seriously alter the rate of the conversion. On the other hand, there are a few processes that are in fact carried out, so far as it is now known, by only a single species, and elimination of that species could have serious consequences. Examples of this are the nitrification process, in which ammonium is converted to nitrate, and the nodulation of leguminous plants, for which the bacteria are reasonably specific according to the leguminous host (Alexander, 1980).

The nitrification process is one of the best indicators of pH stress because the responsible organisms, presumably largely autotrophic bacteria, are sensitive both in culture and in nature to increasing acidity (Dancer et al., 1973). Although nitrification will sometimes occur at pH values below 5.0, characteristically the rate decreases with increasing acidity and often is undetectable much below pH 4.5. Limited data suggest that the process of sulfate reduction to sulfide in soil is markedly inhibited below a pH of 6.0 (Connell and Patrick,

1968) and studies of the presumably responsible organisms in culture attest to the inhibition linked with the acid conditions (Alexander, 1980).

Blue-green algae have been found to be absent from acid soils even though there is both adequate moisture and exposure to sunlight. Studies by Wodzinski et al. (1977) attest to the sensitivity of these organisms to acidity. Inhibition of the rates of both CO_2 fixation and nitrogen fixation was noted.

Studies concerned with the acidification of soil by nitrogen fertilizers or sulfur amendments, as well as comparisons of the microbial populations in soils with dissimilar pH values, attest to the sensitivity of bacteria to increasing hydrogen ion concentrations. Characteristically, the numbers of these organisms decline, and not only is the total bacterial community reduced in numbers, but individual physiological groups are also reduced (Alexander, 1980). The actinomycetes (taxonomically considered to be bacteria) also are generally less abundant as the pH decreases, while the relative abundance of fungi increases, possibly due to a lack of competition from other heterotrophs (Dancer et al., 1973). The pH of soil not only influences the microbial community at large but also those specialized populations that colonize the root surfaces (Alexander, 1980).

It is difficult to make generalizations concerning the effects of soil acidification on microorganisms. Many microbial processes that are important for plant growth are clearly suppressed as the pH declines; however, the inhibition noted in one soil at a given pH may not be noted at the same pH in another soil (Alexander, 1980). The capacity of some microorganisms to become acclimated to changes in pH suggests the need to study this phenomenon using environments that have been maintained at different pH values for some time. Typically the studies have been done with soils maintained only for short periods at the greater acidity (Alexander, 1980). The consequences of increased acidity in the subterranean ecosystem are totally unclear.

Adding nitrate and other forms of nitrogen from the atmosphere to ecosystems is an integral function of the terrestrial nitrogen cycle. Higher plants and microorganisms can assimilate the inorganic forms rapidly. The contribution of inorganic nitrogen in wet precipitation (rain plus snow) is usually equivalent to only a few percent of the total nitrogen assimilated annually by plants in terrestrial ecosystems; however, total nitrogen contributions, including organic nitrogen, in bulk precipitation (rainfall plus dry fallout) can be significant, especially in unfertilized natural systems.

Atmospheric contributions of nitrate can range from less than 0.1 kg N/ha/yr in the Northwest (Fredericksen, 1972) to 4.9 kg N/ha/yr in the eastern United States (Henderson and Harris, 1975; Likens et al., 1970). Inorganic nitrogen (ammonia-N plus nitrate-N) additions in wet precipitation ranged from less than 0.5 kg/ha/yr to more than 3.5 kg/ha/yr in Junge's study (1958) of rainfall over the United States. On the other hand, total nitrogen loads in bulk precipitation range from less than 5 kg/ha/yr in desert regions of the West to more than

30 kg/ha/yr near barnyards in the Midwest. Total contributions of nitrogen from the atmosphere commonly range from about 10 to 20 kg N/ha/yr for most of the United States (National Research Council, 1978).

In comparison, rates of annual uptake by plants range from 11 to 125 kg N/ha/yr in ecosystems selected from several bioclimatic zones (National Research Council, 1978). Since the lowest additions are generally associated with desert areas where rates of uptake by plants are low, and the highest additions usually occur in moist areas where plant uptake is high, the contributions of ammonia and nitrate from rainfall to terrestrial ecosystems are equivalent to about 1 to 10 percent of annual plant uptake. The typical additions of total nitrogen in bulk precipitation, on the other hand, represent from about 8 to 25 percent of the annual plant requirements in eastern deciduous and western coniferous forest ecosystems. Although these comparisons suggest that plant growth in terrestrial ecosystems depends to a significant extent on atmospheric deposition, it is not yet possible to estimate the importance of these contributions by comparing them with the biological fixation and mineralization of nitrogen in the soil. In nutrient-impoverished ecosystems, such as badly eroded abandoned croplands or soils subjected to prolonged leaching by acidic precipitation, nitrogen additions from atmospheric depositions are certainly important to biological productivity. In largely unperturbed forests, recycled nitrogen from the soil organic pool is the chief source of nitrogen for plants, but nitrogen to support increased production must come either from biological fixation or from atmospheric contributions. It seems possible, therefore, that man-generated contributions could play a significant ecological role in a relatively large portion of the forested areas near industrialized regions (National Research Council, 1978).

Sulfur, like nitrogen, is essential for optimal plant growth. Plants usually obtain sulfur from the soil in the form of sulfate. The amount of mineral sulfur in soils is usually low and its release from organic matter during microbial decomposition is a major source for plants (Donahue et al., 1977). Another major source is the wet and dry deposition of atmospheric sulfur (Brady, 1974; Donahue et al., 1977; Jones, 1975).

In agricultural soils crop residues, manure, irrigation water, and fertilizers and soil amendments are important sources of sulfur. The amounts of sulfur entering the soil system from atmospheric sources is dependent on proximity to industrial areas, the sea coast, and marshlands. The prevailing winds and the amount of precipitation in a given region are also important (Halsteand and Rennie, 1977). Near fossil-fueled power plants and industrial installations the amount of sulfur in precipitation may be as much as 150 pounds per acre (168 kg/ha) or more (Jones, 1975). By contrast, in rural areas, based on the equal distribution of sulfur oxide emissions over the coterminous states, the amount of sulfur in precipitation is generally well below the average 15 pounds per acre (17 kg/ha). Approximately 5 to 7 pounds per acre (7 to 8 kg/ha) per year were reported for Oregon in 1966 (Jones, 1975). Shinn and Lynn (1979) have estimated that in the northeastern United States, the area where precipitation is most acidic, approximately 5 x 10^6 tons of sulfate per year is removed by rain

(Brady, 1974). Hoeft et al. (1972) estimated the overall average sulfur as sulfate deposition at 26 pounds of sulfur/acre per year (30 kg S/ha per year). Estimates for rural areas were 14 pounds of sulfur per acre per year (16 kg/ha/yr). Approximately 40 to 50 percent of the sulfur additions occurred from November to February. Tabatabai and Laflin (1976) found that SO_4-S deposition in Iowa was greatest in fall and winter when precipitation was low. They also estimated that the additions of sulfur by precipitation were the same for Ames in 1976 as were reported for 1923, approximately 15 lbs/acre. The average annual additions of sulfur by precipitation were similar to that reported for rural Wisconsin by Hoeft et al. (1972)

Experimental data have shown that even though plants are supplied with adequate soil sulfate they can absorb 25 to 35 percent of their sulfur from the atmosphere (Brady, 1974). Particularly if the soil sulfur is low and atmosphere sulfur high, most of the sulfur required by the plant can come from the atmosphere (Brady, 1974). Atmospheric sulfur would be of benefit chiefly to plants growing on lands with a low sulfur content (Brezonik, 1975).

Tree species vary in their ability to utilize sulfur. Nitrogen and sulfur are biochemically associated in plant proteins, therefore, a close relationship exists between the two in plants. Apparently, nitrogen is only taken up at the rate at which sulfur is available. Protein formation is, therefore, limited by the amount of sulfur available (Turner and Lambert, 1980). Conifers accumulate as sulfate any sulfur beyond the amount required to balance the available nitrogen. Protein formation proceeds at the rate at which nitrogen becomes available. Trees are not injured when sulfur is applied as sulfate rather than SO_2 (Turner and Lambert, 1980).

When discussing the effects of acidic precipitation, or the effects of sulfates or nitrates on soils, a distinction should be made between managed and unmanaged soils. There appears to be general agreement that managed agricultural soils are less susceptible to the influences of acidic precipitation than are unmanaged forest or rangeland soils. On managed soils more than adequate amounts of lime are used to counteract the acidifying effects of fertilizers in agricultural soils. Ammonium fertilizers, usually as ammonium sulfate [$(NH_4)_2SO_4$] or ammonium nitrate, (NH_4NO_3) are oxidized by bacteria to form sulfate (SO_4^{2-}) and/or nitrate (NO_3^-) and hydrogen ions (H^+) (Brady, 1974; Donahue et al., 1977). The release of hydrogen ions into the soil causes the soil to become acidified. Hydrogen ions are also released into the soil when plants take up mineral nutrients. Hence, substances (notably various complexes of ammonium and sulfate ions), although of neutral pH, or nearly so, are acidifying in their effects when they are taken up by plants or animals. Thus, the concept of "acidifying precipitation" must be added to the concept of "acid precipitation."

The acidifying effects of fertilization or acidic precipitation is countered in managed soils through the use of lime. Liming tends to raise the pH and thereby eliminate most major problems associated with acidic soils (Donahue et al., 1977; Likens et al., 1977). Costs of liming all natural soils would be prohibitive as well as extremely difficult to carry out.

Precipitation may add many chemicals to terrestrial, aquatic, and agricultural ecosystems. In addition to sulfur and nitrogen, phosphorus and potassium are biologically most important because they often are in limited supply in the soil (Likens et al. 1977). Other chemicals of varying biological importance and varying concentration found in precipitation over North America are the following: chlorine, sodium, calcium, magnesium, iron, nickel, copper, zinc, cadmium, lead, manganese, (Beamish, 1976; Brezonik, 1975; Hutchinson and Whitby, 1976) mercury, (Brezonik, 1975) and cobalt (Hutchinson and Whitby, 1976). Rain over Britain and the Netherlands, according to Gorham, (1976) contained the following elements in addition to those reported for North American precipitation: aluminum, arsenic, beryllium, cerium, chromium, cesium, antimony, scandium, selenium, thorium, and vanadium. Again it is obvious that many of these elements will be found in precipitation in highly industrialized areas and will not be of biological importance until they enter an ecosystem where they may come into contact with some form of life, as in the case of heavy metals in the waters and soils near Sudbury, Ontario. Of chemical elements found in precipitation, magnesium, iron, copper, zinc, and manganese are essential in small amounts for the growth of plants; however, at high concentrations these elements, as well as the other heavy metals, can be toxic to plants and animals. Furthermore, the acidity of precipitation can affect the solubility, mobility, and toxicity of these elements to the foliage or roots of plants and to animals or microorganisms that may ingest or decompose these plants.

Wiklander (1979) has pointed out that based on the ion exchange theory, ion exchange experiments, and the leaching of soil samples, the following conclusions can be drawn about the acidifying effect on soils through the atmospheric deposition of mineral acids.

1. At a soil pH > 6.0 acids are fully neutralized by decomposition of $CaCO_3$ and other unstable minerals and by cation exchange.
2. At soil pH F 5.5 the efficiency of the proton to decompose minerals and to replace exchangeable Ca^{2+}, Mg^{2+}, K^+, and Na^+ decreases with the soil pH. Consequently, the acidifying effect of mineral acids on soils decreases, but the effect on the runoff water increases in the very acid soils.
3. Salts of CA^{2+}, Mg^{2+}, K^+, and NH_4^+ in the precipitation counteract the absorption of protons and, in that way, the decrease of the base saturation. A proportion of the acids percolate through the soil and acidify the runoff.

The sensitivity of various soils to acidic precipitation depends on the soil buffer capacity and on the soil pH. Noncalcareous sandy soils with pH J 5 are the most sensitive to acidification; however, acidic soils would be most likely to release aluminum.

Very acid soils are less sensitive to further acidification because they are already adjusted by soil formation to acidity and are therefore more stable. In these soils easily weatherable minerals have disappeared, base saturation is low, and the pH of the soil may be less than that of precipitation. The low nutrient level is a crucial factor which limits productivity in these soils. Even a slight decrease in nutrient status by leaching may have a detrimental effect on plant yield (Wiklander, 1979). Fertilization appears to be the only preventive measure.

In properly managed cultivated soils, acidic precipitation should cause only a slight increase in the lime requirement, with the cost compensated for by the supply of sulfur, nitrogen, magnesium, potassium, and calcium made available to plants (Wiklander, 1979).

11.3.2.2 Effects on Vegetation--The atmosphere, as well as the soil, is a source of nutrients for plants. Chemical elements reach the plant surface via wet and dry deposition. Nitrates and sulfates are not the only components of precipitation falling onto the plant surface. Other chemical elements (cadmium, lead, zinc, manganese), at least partially soluble in water, are deposited on the surface of vegetation and may be assimilated by it, usually through the leaves. An average raindrop deposited on trees in a typical forest washes over three tiers of foliage before it reaches the soil. The effects of acidic precipitation may be beneficial or deleterious depending on its chemical composition, the species of plant on which it is deposited, and the physiological condition and maturity of the plant (Galloway and Cowling, 1978). Substances accumulated on the leaf surfaces strongly influence the chemical composition of precipitation not only at the leaf surface, but also when it reaches the forest floor. The chemistry of precipitation reaching the forest floor is considerably different from that collected above the forest canopy or a ground level where the canopy has no influence (Lindberg et al., 1979). Except for the hydrogen ion (H^+) the mean concentrations of all elements (lead, manganese, zinc and cadmium) studied in the Walker Branch Watershed in Tennessee found by Lindberg et al. (1979) to be present in greater amounts in the throughfall than in incident rain. The presence of trace elements was more variable than that of the sulfate and hydrogen ions. Throughfall with a pH 4.5 appeared to be a more dilute solution of sulfuric acid than rain not influenced by the forest canopy. The solution was found to contain a relatively higher concentration of alkaline earth salts of sulfate and nitrate as well as a somewhat higher concentration of trace elements (Lindberg et al., 1979).

Lee and Weber (1980) studied the effects of sulfuric acid rain on two model hardwood forests. The experiment, conducted under controlled field conditions, consisted of the application of simulated sulfuric acid rain (pH values of 3.0, 3.5, and 4.0), and a control rain of pH 5.6 to the two model forest ecosystems for a duration of 3 and 1/2 years. Rainfall applications approximated the annual amounts of areas in which sugar maple and red alder communities normally occur.

In evaluating the results of the study, the authors conclude that a well developed forest canopy and litter layer can increase the pH and concentration of bases (i.e., calcium and magnesium) in rainwater. Such conditions would tend to decrease the acidification rate of forest soils by acid rain. However, as bases are continually leached from the soil column these cations could eventually be lost from the ecosystem and unavailable to influence the acidification reactions. Changes in the ionic and pH balance of forest systems may impact the productivity of forests through acidity-induced changes in the nutrient cycling process, decomposition, reproduction, tree growth, and the structure of forest systems.

The additions of hydrogen, sulfate and nitrate ions to soil and plant systems have both positive and negative effects. It has generally been assumed that the free hydrogen ion concentration in acidic precipitation is the component that is most likely to cause direct, harmful effects on vegetation (Jacobson, 1980). Experimental studies support this assumption; however, to date, there are no confirmed reports of exposure to ambient acidic precipitation causing foliar symptoms on field grown vegetation in the continental United States (Jacobson, 1980) and Canada (Linzon personal communication).

11.3.2.2.1 Direct effects on vegetation. Hydrogen ion concentrations equivalent to that measured in more acidic rain events ($\leq$ pH 3.0) have been observed experimentally to cause tissue injury in the form of necrotic lesions to a wide variety of plant species under greenhouse and laboratory conditions. This visible injury has been reported as occurring between pH 3.0 and 3.6 (Shriner, 1980). The various types of direct effects which have been reported are shown in Table 11-7. Such effects must be interpreted with caution because the growth and morphology of leaves on plants grown in greenhouses frequently are atypical of field conditions (Shriner, 1980).

Small necrotic lesions, the most common form of direct injury, appear to be the result of the collection and retention of water on plant surfaces and the subsequent evaporation of these water droplets once a lesion occurs. The depression formed by the lesion further enhances the collection of water. A large percentage of the leaf area may exhibit lesions after repeated exposures to simulated acid rain at pH concentrations of 3.1, 2.7, 2.5 and 2.3 (Evans et al., 1977a, 1977b). In leaves injured by simulated acidic rain, collapse and distortion of epidermal cells on the upper surface is frequently followed by injury to the palisade cells and ultimately both leaf surfaces are affected (Evans et al., 1977b). Evans et al. (1978) using six clones of Populus spp. hybrids found that leaves that had just reached full expansion were more sensitive to simulated acid rain at pH 3.4, 3.1, 2.9, and 2.7 than were unexpanded or those which were fully expanded. On two of the clones, gall formation due to abnormal cell proliferation and enlargement occurred. Other effects attributed to simulated acid rain include the modification of the leaf surface, e.g. epicuticular waxes, and alteration of physiological processes such as carbon fixation and allocation.

Lee et al. (1980) studied the effects of simulated acidic precipitation on crops. Depending on the crop studied, they reported positive, negative or no effects on crop yield when exposed to sulfuric acid rain at pH values of 3.0, 3.5 and 4.0 when compared to crops exposed to a control rain of pH 5.6. The yield of tomatoes, green peppers, strawberries, alfalfa, orchard grass and timothy were stimulated. Yields of radishes, carrots, mustard greens and broccoli were inhibited. Potatoes were ambiguously affected except at pH 3.0 where

TABLE 11-7. TYPES OF DIRECT, VISIBLE INJURY REPORTED IN RESPONSE TO ACIDIC WET DISPOSITION

Injury Type	Species	pH Range	Reference	Remarks
Pitting, curl shortening, death	Yellow birch	2.3-4.7	Wood and Bormann (1974)	
1-mm necrotic lesions, premature abscission	Kidney bean, soybean, loblolly pine, E. white pine, willow oak	3.2	Shriner et al. (1974)	
Cuticular erosion	Willow oak	3.2	Shriner (1978a) Lang, et al. (1978)	
Chlorosis	Sunflower, bean	2.3-5.7	Evans et al. (1977)	
(A) small, shallow circular depressions: slight chlorosis	Sunflower, bean	2.7	Evans et al. (1977)	More frequent near veins. (A) - (D) represent sequential stages of lesion development, through time, up to 72 h (one 6-min rain event daily for 3 d)
(B) larger lesions, chlorosis always present palisade collapse	Sunflower, bean	2.7	Evans et al. (1977)	
(C) 1-mm necrotic lesions general distortion	Sunflower, bean	2.7	Evans et al. (1977)	
(D) 2-mm bifacial necrosis due to coalescence of smaller lesions, total tissue collapse.	Sunflower, bean	2.7	Evans et al. (1977)	
Wrinkled leaves, excessive adventitious budding, premature abscission	Bean	1.5-3.0	Ferenbaugh (1976)	

TABLE 11-7 (Continued).

Injury Type	Species	pH Range	Reference	Remarks
Incipient bronzed spot	Bean	2.0-3.0	Hindawi et al. (1980)	After first few hours
Bifacial necrotic pitting	Bean	2.0-3.0	Hindawi et al. (1980)	After 24 h (reported pooling of drops = more injury)
Necrotic lesions, premature abscission	E. white pine, scotch pine, spinach, sunflower, bean	2.6-3.4	Jacobsen and van Leuken (1977)	Injury associated with droplet location within 24-48 h.
Marginal and tip necrosis	Bean, poplar, soybean, ash birch, corn, wheat	Submicron H_2SO_4 aerosol	Lang et al. (1978)	
Galls, hypertrophy, hyperplasia	Hybrid poplar	2.7-3.4	Evans et al. (1978)	
Dead leaf cells	Soybean	3.1	Irving (1979)	
Necrotic lesions	Citrus	0.5-2.0	Heagle et al. (1978)	

Shriner (1980).

their yield as well as that of beets was inhibited. Visible injury of tomatoes could possibly have decreased their market value. In sweet corn, stem and leaf production was stimulated, but no statistically significant effects on yield were observed for 15 other crops. Results suggest that the possibility of yield's being affected by acid rain depends on the portion of the plant being utilized as well as the species. Plants were regularly examined for foliar injury associated with acid rain. Of the 35 cultivars examined, the foliage of 31 was injured at pH 3.0; 28 at pH 3.5; and 5 at pH 4.0. Foliar injury was not generally related to effects on yield. However, foliar injury of swiss chard, mustard greens, and spinach was severe enough to adversely affect marketability. These results are from a single growing season and therefore considered to be preliminary.

Studies indicate that wet deposition of acidic or acidifying substances may result in a range of direct or indirect effects on vegetation. Environmental conditions before, during and after a precipitation event affect the responses of vegetation. Nutrient status of the soil, plant nutrient requirements, plant sensitivity and growth stage and the total loading or deposition of critical ions e.g. H^+, NO_3^- and SO_4^- all play a role in determining vegetational response to acidic precipitation.

Wettability of leaves appears to be an important factor in the response of plants to acid deposition. This has been demonstrated in the work of Evans et al. (1977), Jacobson and Van Leuken (1977), and Shriner (1978a), who variously report a threshold of between pH 3.1 and 3.5 for development of foliar lesions on beans. The cultivars of Phaseolus vulgaris L. used in the above studies are all relatively non-waxy and therefore fairly easily wettable. By comparison, studies with the very waxy leaves of citrus (Heagle et al., 1978) reported a threshold for visible symptoms to be near pH 2.0. Waxy leaves apparently minimize the contact time for the acid solutions, thus accounting for the <400X increase in H^+ ion concentration required to induce visible injury. Table 11-8 summarizes the thresholds, including range, species sensitivity, concentration, and time, for visible injury associated with experimental studies of wet deposition of acidic substances.

Leaching of chemical elements from exposed plant surfaces is an important effect rain, fog, mist, and dew have on vegetation. Substances leached include a great diversity of materials. All of the essential minerals, amino acids, carbohydrate growth regulators, free sugars, pectic substances, organic acids, vitamins, alkaloids, and alleopathic substances are among the materials which have been detected in plant leachates (Tukey, 1970). Many factors influence the quantity and quality of the substances leached from foliage. They include factors associated directly with the plant as well as those associated with the environment. Not only are there differences among species with respect to leaching, but individual differences also exist among individual leaves of the same crop and even the same plant, depending on the physiological age of the leaf. Young, actively growing tissues are relatively immune to leaching of mineral nutrients and carbohydrates, while mature tissue which is approaching

TABLE 11-8. THRESHOLDS FOR VISIBLE INJURY AND GROWTH EFFECTS ASSOCIATED WITH EXPERIMENTAL STUDIES OF WET DEPOSITION OF ACIDIC SUBSTANCES (AFTER JACOBSON, 1980a, b)

Effect	Species	Threshold[1]	Reference	Remarks
Foliar lesions, decrease in growth	Yellow birch	pH 3.1	Wood and Bormann (1974)	greenhouse
Foliar aberrations, decrease in growth	Bean	pH 2.5	Ferenbaugh (1976)	greenhouse
Foliar lesions	Bean, sunflower	pH 3.1	Evans et al. (1977a)	greenhouse
Foliar lesions	Bean	pH 3.2	Shriner (1978a)	greenhouse
Foliar lesions	Hybrid poplar	pH 3.4	Evans et al. (1978)	greenhouse
Foliar lesions	Sunflower	pH 3.4	Jocobson and Van Leuken (1977)	greenhouse
Foliar symptons, no reduced growth	Soybean	pH 3.0	Jacobson (1980b)	greenhouse
Increased growth, increased/decreased nutrient content	Lettuce	pH 3.0, 3.2	Jacobson (1980b)	greenhouse (varied with $SO_4^{=}$ & NO_3^{-})
Reduced growth	Pinto bean	pH 3.1	Jacobson (1980b)	greenhouse
Reduced yield	Pinto bean	pH 2.7		
Reduced growth	Soybean	pH 3.1		
Reduced yield	Soybean	pH 2.5		

TABLE 11-8 (Continued).

Effect	Species	Threshold[1]	Reference	Remarks
Increased yield	Soybean	pH 3.1		
Foliar symptoms	Tomato	pH 3.0	Jacobson (1980b)	greenhouse
Reduced growth	Tomato	pH 3.0		
Reduced yield	Tomato	pH 3.0		
Reduced quality	Tomato	pH 3.0		
No foliar symptoms, or effects on growth	Soybean	pH 3.1	Irving (1979)	field
No foliar symptoms, but	Soybean	pH 2.8	Jacobsen (1980b)	field, low ozone
a) decreased growth, yield	Soybean	pH 2.8		field, high ozone
b) increased yield	Soybean	pH 2.8		field, low ozone
No effect on growth, yield	Tomato	pH 3.0	Jacobson (1980b)	field
Reduced quality	Tomato	pH 3.0		field

[1] Highest pH to elicit a negative response, or lowest pH to elicit a positive response

Shriner (1980).

senescence is very susceptible. The stage of plant development, temperature, and rainwater falling on foliage and running down plant stems or tree bark influences leaching. Rainwater, which naturally has a pH of about 5.6, washing over vegetation may become enriched with substances leached from the tissues (Nihlgard, 1970).

Leaching of organic and inorganic materials from vegetation to the soil is part of the normal functioning of terrestrial ecosystems. The nutrient flow from one component of the ecosystem to another is an important phase of nutrient cycling (Comerford and White, 1977; Eaton et al., 1973). Plant leachates have an effect upon soil texture, aeration, permeability, and exchange capacity. Leachates, by influencing the number and behavior of soil microorganisms, affect soil-forming processes, soil fertility, and susceptibility or immunity of plants to soil pests and plant-chemical interactions (Tukey, 1970).

It has been demonstrated under experimental conditions that precipitation of increased acidity can increase the leaching of various cations and organic carbon from the tree canopy (Abrahamsen et al., 1976; Wood and Bormann, 1975). Foliar losses of potassium, magnesium, and calcium from bean plants and maple seedlings were found to increase as the acidity of an artificial mist was increased. Below a pH of 3.0 tissue damage occurred; however, significant increases in leaching were measured at pH 3.3 and 4.0 with no observable tissue damage (Wood and Bormann, 1975). Hindawi et al. (1980) also noted that as the acidity of acid mist increased so did the foliar leaching of nitrogen, calcium, phosphorous, and magnesium. Potassium concentrations were not affected, while the concentration of sulfur increased. Abrahamsen and Dollard (1979), in experiments using Norway spruce (Picea abies L. Karst), observed that despite increased leaching under the most acid treatment, there was no evidence of change in the foliar cation content. Wood and Bormann (1977), using Eastern white pine (Pinus strobus L.), also noted no significant changes in calcium, magnesium or potassium content of needles. Tukey (1970) states that increased leaching of nutrients from foliage can accelerate nutrient uptake by plants. No injury will occur to the plants as long as roots can absorb nutrients to replace those being leached; however, injury could occur if nutrients are in short supply. To date, the effects, if any, of the increased leaching of substances from vegetation by acidic precipitation remain unclear.

Some experimental evidence suggests that acidic solutions affect the chlorophyll content of leaves and the rate of photosynthesis. Sheridan and Rosenstreter (1973) reported marked reduction of photosynthesis in a moss exposed to increasing H^+ ion concentrations. Sheridan and Rosenstreter (1973), Ferenbaugh (1976), and Hindawi et al. (1980) reported reduced chlorophyll content as a result of tissue exposure to acid solutions. In the case of Ferenbaugh (1976), however, the significant reductions in chlorophyll in the leaves of Phaseolus vulgaris at pH 2.0 were associated with large areas of necrosis. A significant aspect of this study was the loss of capacity by the plant to produce carbohydrates. The rate of respiration in these plants showed only a slight but significant increase while the rate of photosynthesis at pH 2.0 increased nearly fourfold as determined by oxygen evolution. Ferenbaugh concluded that due to a reduction in biomass accumulation and sugar and starch concentrations, photophosphorylation in the treated plants was in some way being uncoupled by the acidic solutions.

Irving (1979) reported a higher chlorophyll content and an increase in the rate of photosynthesis in field-grown soybeans exposed to simulated rain at pH 3.1. She attributed the increases to improved nutrition due to the sulfur and nitrogen components of the simulated acid rain overcoming any negative effects.

Vegetation is commonly exposed to gaseous phytotoxicants such as ozone and sulfur dioxide at the same time as acidic precipitation. Little information is available upon which to evaluate the potential for determining the effects of the interaction of wet-and dry-deposited pollutants on vegetation. Preliminary studies by Shriner (1978b), Irving (1979), and Jacobson et al. (1980) suggest that interactions may occur. Irving (1979) found that simulated acid precipitation at pH of 3.1 tended to limit the decrease in photosynthesis observed when field -grown soybeans were exposed 17 times during the growing season to 0.19 ppm of SO_2. Shriner (1978b), however, reported no significant interaction between multiple exposure to simulated rain at p 4.0 and four SO_2 exposures (3 ppm peak for 1 hr.) upon the growth of bush beans. Shriner (1978b) also exposed plants to 0.15 ppm ozone (4 3-hour exposures) in between 4 weekly exposures to rainfall of pH 4.0, and observed a significant growth reduction at the time of harvest. Jacobson et al. (1980), using open-top exposure chambers with field-grown soybeans, compared growth and yield between three pH levels of simulated rain (pH 2.8, 3.4, and 4.0) and two levels of ozone (<0.03 and $\leq$0.12 ppm). Results demonstrated that ozone depressed both growth and yield of soybeans with all three rain treatments, but that the depression was greatest with the most acidic rain. Ozone concentrations equal to or greater than those used in the studies are common in most areas of the northeastern United States where acidic deposition is a problem (Jacobson et al., 1980); therefore, the potential for ozone-acidic deposition interaction is great.

Shriner (1978a) studied the effect of acidic precipitation on host-parasite interactions. Simulated acid rain with a pH of 3.2 inhibited the development of bean rust and production of telia (a stage in the rust life cycle) by the oak-leaf rust fungus Cronartium fusiforme. It also inhibited reproduction of root-knot nematodes and inhibited or stimulated development of halo blight of bean seedlings depending on the time in the disease cycle during which the simulated acid rain was applied. The effects which inhibited disease development could result in a net benefit to plant health. Shriner (1976, 1980) also observed that root nodulation by Rhizobium on common beans and soybeans was inhibited by the simulated acid rain, suggesting a potential for reduced nitrogen fixation by legumes so effected.

Plants such as mosses and lichens are particularly sensitive to changes in precipitation chemistry because many of their nutrient requirements are obtained directly through precipitation. These plant forms are typically absent from regions with high chronic SO_2 air pollution and acidic precipitation (Denison et al., 1977; Sheridan and Rosenstreter, 1973). Gorham (1976) and Giddings and Galloway (1976) have written reviews concerning this problem. Most investigations on the effects of air pollution on epiphytes have dealt with gaseous pollutants. Very few studies have considered acidic precipitation. Denison et al. (1977), however, did observe that the nitrogen-fixing ability of the epiphytic lichen Lobaria oregana was reduced when treated with simulated rainfall with a pH of 4.0 and below. Investigations concerning the effects of acidic precipitation on epiphytic microbial populations are very few (Abrahamsen and Dollard, 1979).

Limited fertilization could occur in the bracken fern Pteridium aquilinum under conditions of acidic precipitation (pH and sulfate concentrations) that prevail in the northeastern United States. Evans and Bozzone (1977), using buffered solutions to simulate acidic precipitation, observed that flagellar movement of sperm was reduced at pH levels below 5.8. Fertilization was reduced after exposure to pH's below 4.2. Sporophyte production was also reduced by 50 percent at pH levels below 4.2 when compared to 5.8. Addition of sulfate (86 mM) decreased fertilization at least 50 percent at all pH values observed. In another study, Evans and Bozzone (1978) observed that both sperm motility and fertilization in gametophytes of Pteridium aquilinum were reduced when anions of sulfate, nitrate, and chloride were added to buffered solutions.

Sulfur and nitrogen in precipitation have been shown to play an important role in vegetational response to acidic deposition. Jacobson et al. (1980) investigated the impact of simulated acidic rain on the growth of lettuce at acidities of pH 5.7 and 3.2. At pH 3.2, solutions were compared with $NO_3:SO_4$ mass ratios of 20:1, 2:1, and 1:7.5. The high nitrate at pH 3.2 showed no difference from the treatments controls at pH 5.7 for those growth parameters (root dry weight, apical leaf dry weight) that responded to treatment; however, the results were significantly less than those from the low nitrogen, high sulfur, treatment. These observations suggest that sulfur was possibly a limiting factor in the nutrition of these plants, with the result that the plant response to sulfur overwhelmed the hydrogen ion effect. Other studies also have cited the beneficial effects of simulated acidic precipitation. Irving and Miller (1978) observed that an acidic simulant had a positive effect on productivity of field-grown soybeans as reflected by seed weight. Increased growth was attributed to a fertilizing effect from sulfur and nitrogen delaying senescence. Irving and Miller (1978), in the same study, also exposed soybeans to SO_2 and acidic precipitation. No visible injury was apparent in any of the plots; however, a histological study revealed significant increases in the number of dead mesophyll cells in all plots when compared to the control. The proportion of dead mesophyll cells of plants exposed to acid rain and SO_2 combined was more than additive when compared to the effects of each taken singly. Wood and Bormann (1977) reported an increase in needle length and the weight of seedlings of Eastern white pine with increasing acidity of simulated precipitation where sulfuric and nitric acid were used to acidify the mist. Increased growth was attributed to increased NO_3^- application. Abrahamson and Dollard (1979) also presented data suggesting positive growth responses in forest tree species resulting from nitrogen and sulfur in simulated rain. Simulated acidic precipitation was observed to increase the growth of Scots pine saplings in experiments conducted in Norway. Saplings in plots watered with acid rain of pH 3.0, 2.5, and 2.0 grew more than the control plots. The application of acid rain increased the nitrogen and sulfur content of the needles. As the acidity of the artificial rain was adjusted using sulfuric acid only, the increased growth was probably due to increased nitrogen mineralization and uptake. Turner and Lambert (1980) reported evidence indicating a positive growth response in Monterey pine from the deposition of sulfur in ambient precipitation in Australia.

Acidifying forest soils that are already acid by acidic precipitation or air pollutants is a slow process. Growth effects probably could not be detected for a long time. To identify the possible effects of acidification on poor pine forests, Tamm et al. (1977) conducted experiments using 50 kg and 100 kg of sulfur per hectare as dilute sulfuric acid (0.4 percent) applied annually with and without NPK (nitrogen, phosphorous, potassium) fertilizer. Nitrogen was found to be the limiting factor at both experimental sites. Acidification produced no observable influence on tree growth. Lysimeter and soil incubation experiments conducted at the same time as the experiments described above suggest that even moderate additions of sulfuric acid or sulfur to soil affect soil biological processes, particularly nitrogen turnover. The soil incubation studies indicated that additions of sulfuric acid increased the amount of mineral nitrogen but lowered the amount of nitrate.

Soil fertility may increase as a result of acidic precipitation as nitrate and sulfate ions, common components of chemical fertilizers, are deposited; however, the advantages of such additions are possibly short-lived as depletion of nutrient cations through accelerated leaching could eventually retard growth (Wood, 1975). Laboratory investigations by Overrein (1972) have demonstrated that leaching of potassium, magnesium, and calcium, all important plant nutrients, is accelerated by increased acidity of rain. Field studies in Sweden correlate decreases in soil pH with increased additions of acid (Oden, 1972).

Major uncertainty in estimating effects of acid rain on forest productivity is the capacity of forest soils to buffer against leaching by hydrogen ions. Forest canopies have been found to filter 90 percent of the hydrogen ions from rain (pH 4.0) falling on the landscape during the growing season (Eaton et al., 1973). As a result, solutions reaching the forest floor are less acidic (pH 5.0). Mayer and Ulrich (1977), however, point out that their studies suggest that for most elements the addition by precipitation (wetfall plus dryfall) to the soil beneath the tree canopy is considerably larger than that by precipitation to the canopy surface as measured by rain gauges on a non-forested area. The leaching of metabolites, mainly from leaf surfaces, and the washing out from leaves, branches, and stems of airborne particles and atmospheric aerosols intercepted by trees from the atmosphere, are suggested as the reason for the mineral increase.

Forest ecosystems are complicated biological organizations. Acidic precipitation will cause some components within the ecosystem to respond even though it is not possible at present to evaluate the changes that occur. The impact of the changes on the ecosystem can only be determined with certainty after the passage of a long period of time.

11.3.2.3 Effects on Human Health--One effect of acidification that is potentially of concern to human health is the possible contamination by toxic metals of edible fish and of water supplies. Studies in Sweden (Landner and Larsson, 1975; Turk and Peters, 1977), Canada (Tomlinson, 1979; Brouzes et al., 1977), and the United States (Tomlinson, 1979) have revealed high mercury concentrations in fish from acidified regions. Methylation of mercury to monomethyl mercury occurs at low pH while dimethyl mercury forms at higher pH (Fageström and Jernelöv, 1972). Monomethyl mercury in the water passing through the gills of fish reacts with thiol groups in the hemoglobin of the blood and is then transferred to the muscle. Methyl mercury is eliminated very slowly from fish; therefore, it accumulates with age.

Tomlinson (1979) reports that in the Bell River area of Canada precipitation is the source of mercury. Both methyl mercury and inorganic mercury were found in precipitation. The source of mercury in snow and rain was not known at the time of the study.

Zinc, manganese, and aluminum concentrations also increase as the acidity of lakes increases (Schofield, 1976b). The ingestion of fish contaminated by these metals is a distinct possibility.

Another human health aspect is the possibility that, as drinking-water reservoirs acidify, owing to acidic precipitation, the increased concentrations of metals may exceed the public-health limits. The increased metal concentrations in drinking water are caused by increased watershed weathering and, more important, increased leaching of metals from household plumbing. Indeed, in New York State, water from the Hinckley Reservoir has acidified to such an extent that "lead concentrations in water in contact with household plumbing systems exceed the maximum levels for human use recommended by the New York State Department of Health" (Turk and Peters, 1977). The lead and copper concentrations in pipes which have stood over night (U) and those in which the water was used (F) are depicted in Table 11-9.

11.3.2.4 Effects of Acidic Precipitation on Materials--Acidic precipitation can damage the abiotic as well as the biotic components of an ecosystem. Of particular concern in this section are the deteriorative effects of acidic precipitation on materials and cultural artifacts of manmade ecosystems. At present in most areas, the dominant factor in the formation of acidic precipitation is sulfur, usually as sulfur dioxide (Cowling and Dochinger, 1978; Likens, 1976). Because of this fact, it is difficult to isolate the effect of acidic precipitation from changes induced by sulfur pollution in general. (The effects of sulfur oxides on materials are discussed in Chapter 10.) High acidity promotes corrosion because the hydrogen ions act as a sink for the electrons liberated during the critical corrosion process (Nriagu, 1978). Precipitation as rain affects corrosion by forming a layer of moisture on the surface

TABLE 11-9. LEAD AND COPPER CONCENTRATION AND pH OF WATER FROM PIPES CARRYING OUTFLOW FROM HINCKLEY BASIN AND HANNS AND STEELE CREEK BASIN, NEAR AMSTERDAM, NEW YORK

Collection site and date	Pipe condition[1]	Copper (μg/l)	Lead (μg/l)	pH
Hinckley Dam				
Nov. 21, 1974	U	600	66	---
Nov. 21, 1974	F	20	2	7.4
Nov. 7, 1974	U	460	40	6.3
Nov.7, 1974	F	37	6	6.3
Oct. 1, 1974	U	570	52	6.8
Oct. 1, 1974	F	30	5	7.1
Aug. 15, 1974	U	760	88	6.3
Aug. 15, 1974	F	40	2	6.3
Amsterdam				
Jan. 6, 1975	U	2900	240	4.5
Jan. 6, 1975	F	80	3	5.0

[1] U, unflushed, (water stands in pipes all night); F, flushed
From Turk and Peters (1977).

of the material and by adding hydrogen (H^+) and sulfate (SO_2^{2-}) ions as corrosion stimulators. Rain also washes out the sulfates deposited during dry deposition and thus serves a useful function by removing the sulfate and stopping corrosion (Kucera, 1976). Rain plays a critical role in the corrosive process because in areas where dry deposition predominates the washing effect is greatest, while in areas where the dry and wet deposition processes are roughly equal, the corrosive effect is greater (Kucera, 1976). The corrosion effect, particularly of certain metals, in areas where the pH of precipitation is very low may be greatly enhanced by that precipitation (Kucera, 1976). In a Swedish study the sulfur content of precipitation, expressed as meq/m^2 per year, was found to correlate closely with the corrosion rate of steel. The metals most likely to be corroded by precipitation with a low pH are those whose corrosion resistance may be ascribed to a protective layer of basic carbonates, sulfates, or oxides, as used on zinc or copper. The decrease in pH of rainwater to 4.0 or lower may accelerate the dissolution of the protective coatings (Kucera, 1976).

Materials reported to be affected by acidic precipitation, in addition to steel, are: copper materials, linseed oil, alkyd paints on wood, antirust paints on steel, limestone, sandstone, concrete, and both cement-lime and lime plaster (Cowling and Dochinger, 1978).

Stone is one of the oldest building materials used by man and has traditionally been considered one of the most durable because structures such as the pyramids, which have survived since antiquity, are made of stone. What is usually forgotten is that the structures built with stone that was not durable have long since disappeared (Sereda, 1977).

Atmospheric sulphur compounds (mainly sulfur dioxide, with subsidiary amounts of sulfur trioxide and ammonium sulfate) react with the carbonates in limestone and dolomites, calcareous sandstone and mortars to form calcium sulfate (gypsum). The results of these reactions are blistering, scaling, and loss of surface cohesion, which in turn induces similar effects in neighboring materials not in themselves susceptible to direct attack (Sereda, 1977).

Sulfates have been implicated by Winkler (1966) as very important in the disintegration of stone. The surface flaking on the Egyptian granite obelisk (Cleopatra's Needle) in Central Park, New York is cited as an example. The deterioration occurred within two years of its erection in 1880.

A classic example of the effects of the changing chemical climate on the stability of stone is the deterioration of the Madonna at Herten Castle, near Recklinghausen, Westphalia in Germany. The sculpture of porous Baumberg sandstone was erected in 1702. Pictures taken of the Madonna in 1908 shows slight to moderate damage during the first 206 years. The features of the Madonna--eyes, nose, mouth and hair--are readily discernable. In pictures taken in 1969 after 267 years, no features are visible (Cowling and Dochinger, 1978).

It is not certain in what form sulfur is absorbed into stone, as a gas (SO_2) forming sulfurous or sulfuric acid or whether it is deposited in rain. Rain and hoarfrost both contain sulfur compounds. Schaffer (1932) compared the sulfate ion in both rain and hoarfrost at Heachingley, Leeds, England in 1932 (Table 11-10) and showed that the content of hoarfrost was approximately 7 times greater than rain. Wet stone surfaces unquestionably increase the condensation or absorption of sulfates. Stonework kept dry and shielded from rain, condensing dew, or hoarfrost will be damaged less by SO_2 pollution than stone surfaces which are exposed (Sereda, 1977).

Acid rain may leach ions from stonework just as acidic runoff and ground water leaches ions from soils or bedrock; however, at the present time it is not possible to attribute the deleterious effects of atmospheric sulfur pollution to specific compounds.

Microbial action can also contribute to the deterioration of stone surfaces. Tiano et al. (1975) isolated large numbers (250 to 20,000 cells per gram) of sulfate-reducing bacteria from the stones of two historical buildings of Florence, Italy. The majority of the bacteria belonged to the genus Thiobacillus. This genus of chemosynthetic aerobic microorganisms oxidizes sulfide, elemental sulfur, and thiosulphate to sulfate to obtain energy (Anderson, 1978). Limestone buildings and particularly mortar used in the construction of brick and stone buildings are particularly susceptible to when Thiobacillus can convert reduced forms of sulfur to sulfuric acid. Sulfate in acidic precipitation as well as other sulfur compounds

TABLE 11-10. COMPOSITION OF RAIN AND HOARFROST AT HEADINGLEY, LEEDS

	Average rain parts per million	Hoarfrost parts per million
Suspended matter	115	4620
Tar	15	158
Ash	28	67
Acidity	1.9	102.9
Sulphur as SO_3	22	148
Sulphur as SO_2	5.7	41.0
Total sulphur	27.7	189.0
Chlorine	7.3	94.6
Nitrogen as NH_3	1.98	8.57
Nitrogen as N_2O_5	0.196	0.0
Nitrogen as albuminoid	0.434	1.618

Schaffer (1932).

deposited in dry deposition could permit the formation of sulfur compounds utilizable by microorganisms. (For more information concerning the effects of sulfur oxides on materials, please consult Chapter 10).

11.4 ASSESSMENT OF SENSITIVE AREAS

11.4.1 Aquatic Ecosystems

Why do some lakes become acidified by acidic precipitation and others not? What determines susceptibility? Are terrestrial ecosystems likely to be susceptible; if so, which ones?

The sensitivity of lakes to acidification is determined by: (1) the acidity of both wet deposition (precipitation) and dry deposition, (2) the hydrology of the lake, (3) the soil system, geology, and canopy effects, (4) the surface water. Given acidic precipitation, the soil system and associated canopy effects are most important. The hydrology of lakes includes the sources, amounts, and pathways of water entering and leaving a lake. The capability of a lake and its drainage basin to neutralize acidic contributions as well as the mineral content of its surface water is largely governed by the composition of the bedrock of the watershed. The chemical weathering of the watershed strongly influences the salinity (ionic composition) and the alkalinity (hardness and softness) of the surface water of a lake (Wetzel, 1975; Wright and Gjessing, 1976; Wright and Henriksen, 1978). The cation exchange capacity and weathering rate of the watershed and the alkalinity of the surface water determine the ability of the system to neutralize the acidity of precipitation.

Lakes vulnerable to acidic precipitation have been shown to have watersheds whose geological composition is highly resistant to chemical weathering (Galloway and Cowling, 1978; Wright and Gjessing, 1976; Wright and Henriksen, 1978). In addition, the watersheds of the vulnerable lakes usually have thin, poor soils and are poorly vegetated. The cation exchange capacity of such soils is low and, therefore, its buffering capacity is low (Schofield, 1979; Wright and Henriksen, 1978).

Wright and Henriksen (1978) point out that the chemistry of Norwegian lakes could be accounted for primarily on the basis of bedrock geology. They examined 155 lakes and observed that 59 of them lay in granite or felsic gneiss basins. Water in these lakes was low in most major ions and had low electrical conductivity. [The fewer the minerals in water the lower its conductivity (Wetzel, 1975).] The waters in the lakes surveyed were "among the softest waters in the world" (Wright and Henriksen, 1978). Sedimentary rocks generally weather readily, whereas igneous rocks are highly resistant. The Adirondacks, as pointed out by Schofield (1975), have granite bedrock with much of the area covered with a mantle of mixed -gneisses. Shallow soils predominate in the area. Thus, areas are susceptible to acidification.

Limestone terrains, on the other hand, are capable of buffering intense concentrations of acids. Glacially derived sediment has been found to be more important than bedrock in assimilating acidic precipitation in the Canadian Shield area (Kramer, 1976). Generally, however, bedrock geology is the best predictor of the sensitivity of aquatic ecosystems to acidic precipitation (Hendrey et al., 1980).

Areas with aquatic ecosystems that have the potential for being sensitive to acidic precipitation are shown in Figure 11-28. In Figure 11-27, the shaded areas on the map indicate that the bedrock is composed of igneous or metamorphic rock while in the unshaded areas it is of calcareous or sedimentary rock. Metamorphic and igneous bedrock weathers slowly; therefore, lakes in areas with this type of bedrock would be expected to be dilute and of low alkalinity [<0.5 meq HCO_3^-/liter (Galloway and Cowling, 1978)]. Galloway and Cowling (1978) verified this hypothesis by compiling alkalinity data. The lakes having low alkalinity existed in regions having igneous and metamorphic rock (Galloway and Cowling, 1978). Hendrey et al. (1980) have developed new bedrock geology maps of the eastern United States for predicting areas which might be impacted by acidic precipitation. The new maps permit much greater resolution for detecting sensitivity than has been previously available for the region.

Henriksen (1979) has developed a lake acidification "indicator model" using pH-calcium and calcium-alkalinity relationships as an indicator for determining decreased surface water

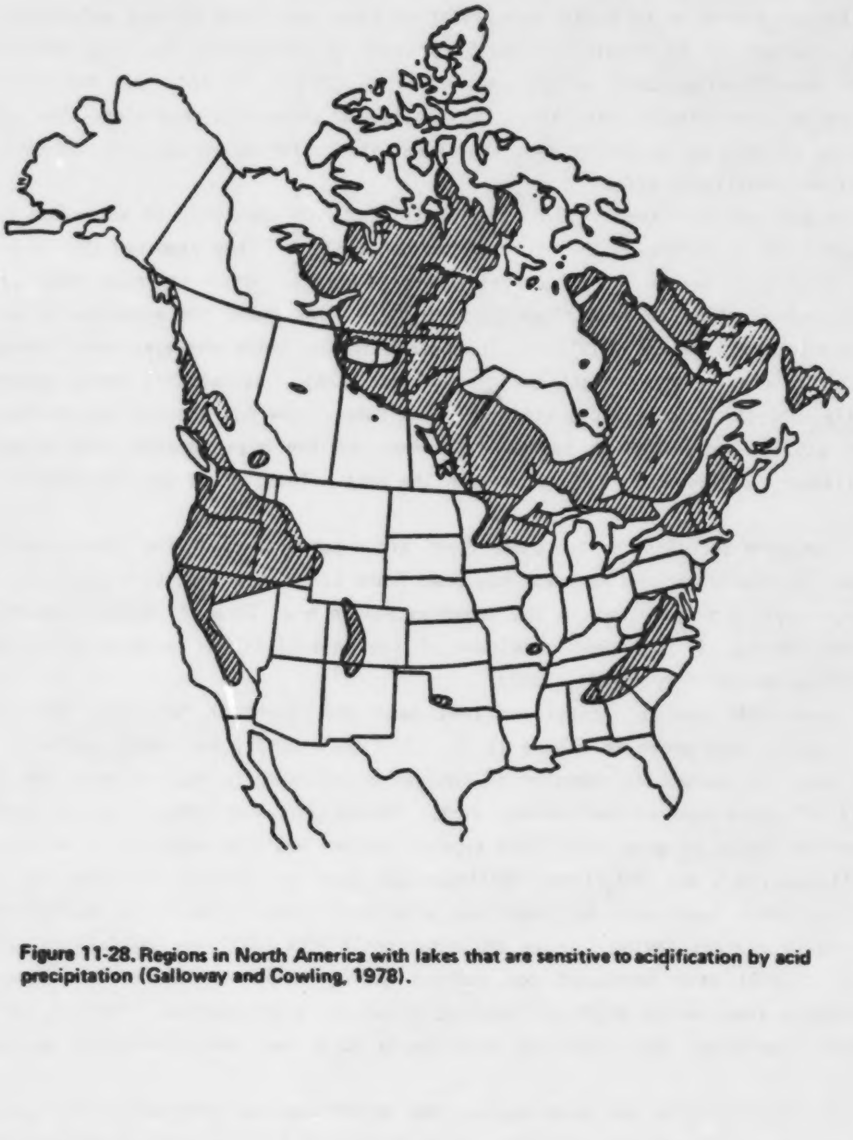

Figure 11-28. Regions in North America with lakes that are sensitive to acidification by acid precipitation (Galloway and Cowling, 1978).

acidification. The indicator is based on the observation that in pristine lake environments (e.g., Northwest Norway or the Experimental Lakes Area in Northwest Ontario, Canada) calcium is accompanied by a proportional amount of bicarbonate because carbonic acid is the primary chemical weathering agent. The pH-calcium relationship found for such regions is thus defined as the reference level for unacidified lakes. Acidified lakes (e.g., Southeast Norway and the Adirondack region) will exhibit lower pH or lower alkalinity than the reference lakes, at comparable calcium levels, due to the replacement of bicarbonate by strong acid anions.

Schofield (1979) has illustrated the use of Henriksen's model with data from Norway, the Adirondacks, and the Experimental Lakes Area of Ontario, Canada. In the acidified lakes sulfate replaces bicarbonate as the major anion present (Figures 11-29 and 11-30) and is derived primarily from precipitation. Since the bicarbonate lost in acidified lakes has been replaced by an equivalent amount of sulfate, the concentration of excess sulfate serves as an index of the amount of acidification that has taken place. Henriksen (1979) compared estimated acidification in Norwegian lakes to the pH and sulfate concentrations in the prevailing precipitation and concluded that significant lake acidification had occurred in areas receiving precipitation with an annual average (volume weighted) pH below 4.6 to 4.7 and sulfate concentrations above 1 mg/l. This approximate threshold of precipitation acidity may be applicable to sensitive lake districts in other regions as well. For reference, the estimated annual bulk deposition sulfate for the acidified lake districts in the Adirondacks and southern Norway are approximately 30 to 60 kg SO_4/ha, as compared with only 5 to 10 kg SO_4/ha in the reference areas of northern Norway and the Experimental Lakes Area in Ontario. A comparison of lake pH with regional sulfate loading levels in Sweden suggests that critical loading levels for sensitive lakes are in the range of 15 to 20 kg SO_4/ha/yr. The amount of precipitation must also be considered since it affects total sulfate additions.

The report by Hendrey et al. (1980) compared pre-1970 data with post-1975 data. A marked decline in both alkalinity and pH of sensitive waters of North Carolina and New Hampshire were tested. In the former, pH and alkalinity have decreased in 80 percent of the streams and in the latter pH has decreased 90 percent since 1949. These areas are predicted to be sensitive by the geological map on the basis of their earlier alkalinity values. Detailed county by county maps of other states in the eastern United States suggest the sensitivity of these regions to acidic precipitation.

Though bedrock geology generally is a good predictor of the susceptibility of an area to acidification due to acidic precipitation, other factors also have an influence. Florida, for example, is underlaid by highly calcareous and phosphate rock, suggesting that acidification of lakes and streams is highly unlikely. Many of the soils, however, (particularly in northern Florida) are very mature, have been highly leached of calcium carbonate, and, as a result, some lakes in which groundwater inflow is minimal have become acidified (Hendrey et al., 1980). Conversely, there are areas in Maine with granitic bedrock where lakes have

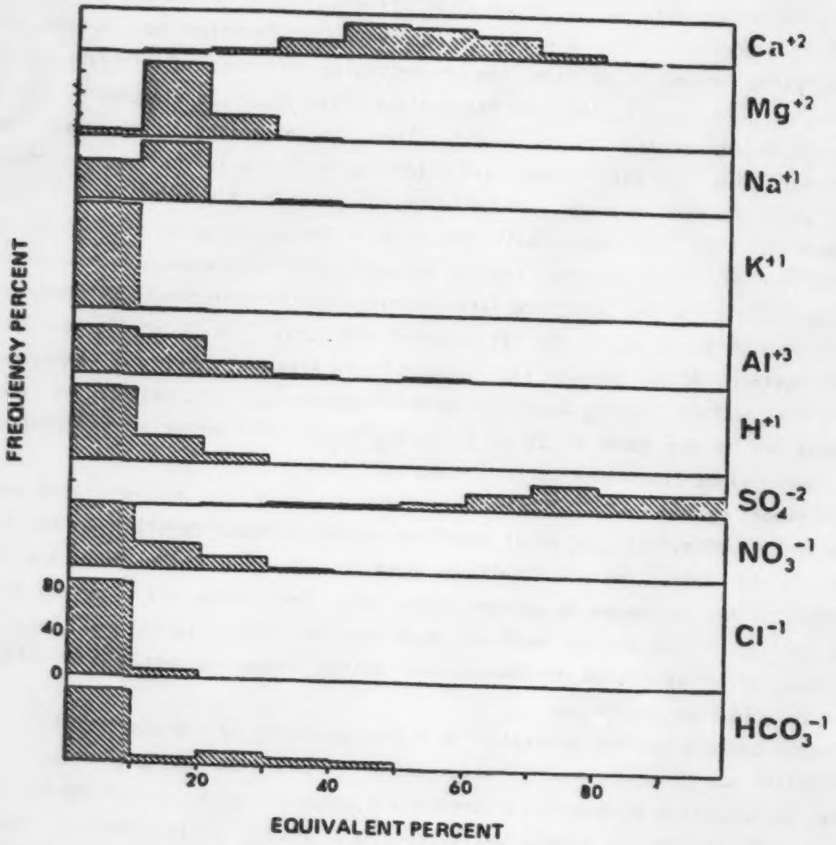

Figure 11-29. Equivalent percent composition of major ions in Adirondack lake surface waters (215 lakes) sampled in June 1975 (Schofield, 1979).

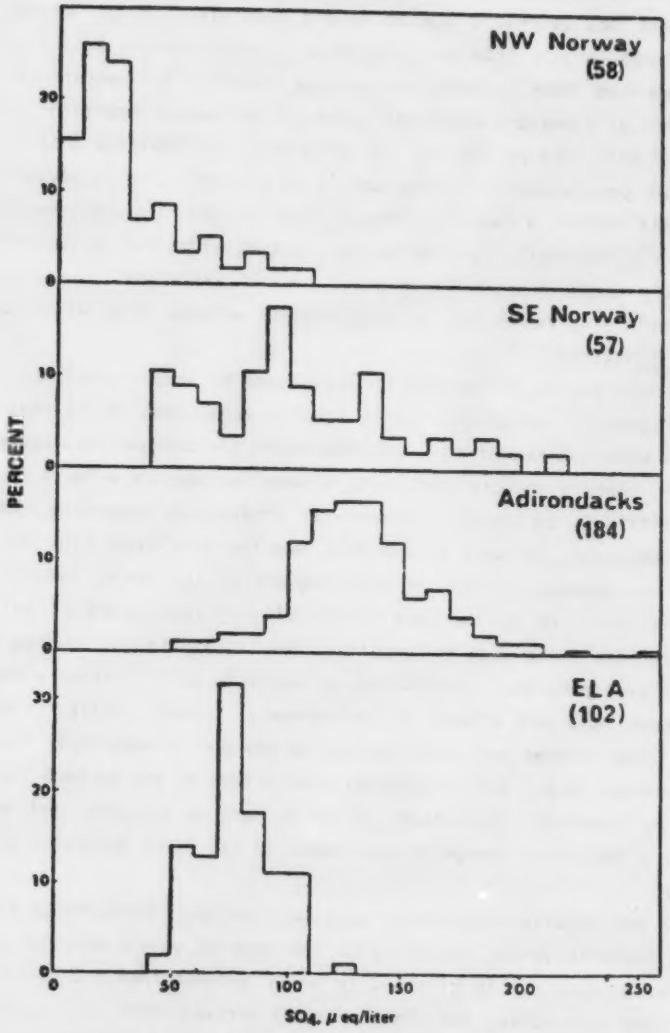

Figure 11-30. Percent frequency distribution of sulfate concentrations in surface water from lakes in sensitive regions (Schofield, 1979). (ELA refers to Experimental Lake Area. Figures in parentheses refer to number of lakes.)

not become acidified, despite receiving precipitation with an average pH of approximately 4.3, because the drainage basins contain lime-bearing till and marine clay (Davis et al., 1978). Small amounts of limestone in a drainage basin exert a strong influence on water quality in terrain which would otherwise be vulnerable to acidification. Soils in Maine in the areas where the pH of lakes has decreased due to acidic precipitation are immature, coarse, and shallow and are derived largely from granitic material and commonly have a low capacity for assimilating hydrogen ions from leachate and surface runoff in lake watersheds (Davis et al., 1978). The occurrence of limestone outcroppings in the Adirondack Mountains of New York state are highly correlated with lake pH levels. The occurrence of limestone apparently counteracts any effects of acidic precipitation. Consequently, when predicting vulnerability of a particular region to acidification, a careful classification of rock mixtures should be made. Rock formations should be classified according to their potential buffering capacity, and the type of soil overlying the formations should be noted. Local variations in bedrock and soils are very important in explaining variations in acidification between lakes within an area.

11.4.2 Terrestrial Ecosystems

Predicting the sensitivity of terrestrial ecosystems to acidic precipitation is much more difficult than for aquatic ecosystems. With aquatic ecosystems it is possible to compare affected ecosystems with unaffected ones and note where the changes have occurred. With terrestrial ecosystems, comparisons are difficult to make because the effects of acidic precipitation have been difficult to detect. Therefore, predictions regarding the sensitivity of terrestrial ecosystems must, as much as possible, use the data which link the two ecosystems, i.e., data on bedrock geology. Since, in most regions of the world, bedrock is not exposed but is covered with soil, it is the sensitivity of different types of soil which must be assessed. Therefore, the first step is to define "sensitivity" as it is used here in relation to soils and acidic precipitation. Sensitivity of soils to acidification alone, though it may be the most important long-term effect, is too narrow a concept. Soils influence the quality of waters in associated streams and lakes and may be changed in ways other than simple pH-base saturation relationships, e.g., microbiological populations of the surface layers, accelerated loss of aluminum by leaching. Therefore, criteria need to be used that would relate soil "sensitivity" to any important change brought about in the local ecosystem by acid precipitation (McFee, 1980).

All soils are not equally susceptible to acidification. Sensitivity to leaching and to loss of buffering capacity varies according to the type of parent material from which a soil is derived. Buffering capacity is greatest in soils derived from sedimentary rocks, especially those containing carbonates, and least in soils derived from hard crystalline rocks such as granites and quartzites (Gorham, 1958). Soil buffering capacity varies widely in different regions of the country (Figure 11-31). Unfortunately, many of the areas now receiving the most acidic precipitation also are those with relatively low natural buffering capacities.

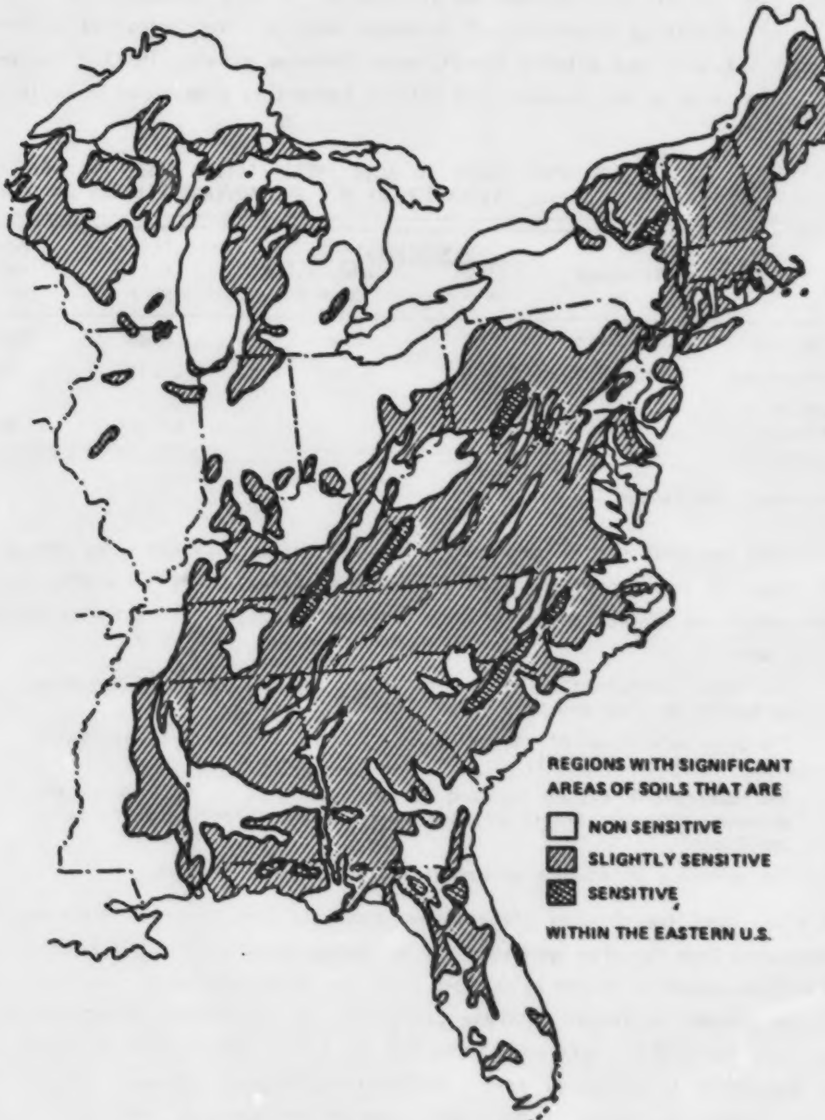
REGIONS WITH SIGNIFICANT AREAS OF SOILS THAT ARE
☐ NON SENSITIVE
▨ SLIGHTLY SENSITIVE
▩ SENSITIVE
WITHIN THE EASTERN U.S.

Figure 11-31. Soils of the eastern United States sensitive to acid rainfall (McFee, 1980).

The buffering capacity of soil depends on mineralogy, texture, structure, organic matter, pH, base saturation, salt content, and soil permeability. Above a pH of 5.5 virtually all of the H^+ ions, irrespective of source, are retained by ion exchange and chemical weathering. Below pH 5.5, the retention of the H^+ ion decreases with the soil pH in a manner determined by the composition of the soil (Donahue et al., 1977). With a successive drop in the soil pH below 5.0, an increasing proportion of hydrogen ions (H^+) and deposited sulfuric acid will pass through the soil and acidify runoff water (Donahue et al., 1977). The sensitivity of different soils based on pH, texture, and calcite content is summarized in Table 11-11.

TABLE 11-11. THE SENSITIVITY TO ACID PRECIPITATION BASED ON: BUFFER CAPACITY AGAINST pH-CHANGE, RETENTION OF H^+, AND ADVERSE EFFECTS ON SOILS

	Calcareous soils	Noncalcareous clays pH > 6	Noncalcareous sandy soils pH > 6	Cultivated soils pH > 5	Acid soils pH < 5
Buffering	Very high	High	Low	High	Moderate
H^+retention	Maximal	Great	Great	Great	Slight
Adverse effects	None	Moderate	Considerable	None - slight	Slight

Reference: Wiklander (1979).

Soils are the most stable component of a terrestrial ecosystem. Any changes which occur in this component would probably have far-reaching effects. McFee (1980) has listed four parameters which are of importance in estimating the sensitivity of soils to acidic precipitation. They are:

1. The total buffering or cation exchange capacity which is provided primarily by clay and soil organic matter.
2. The base saturation of that exchange capacity which can be estimated from the pH of the soil.
3. The management system imposed on the soil; is it cultivated and amended with fertilizers or lime or renewed by flooding or by other additions?
4. The presence or absence of carbonates in the soil profile.

In order that the factors listed above could be used in broad scale mapping of soils, McFee evaluated them for wide applicability and ready availability. In natural soils the most serious effects would be caused by changes in pH by leaching of soil minerals. Susceptibility of soils to changes in either of these categories is most closely associated with the cation exchange capacity (CEC). Soil with a low CEC and a circumneutral pH is likely to have the pH rapidly reduced by an influx of acid. Soils with a high CEC, however, are strongly buffered against pH changes or changes in the composition of the leachate. Acidic soils with a pH near that of acidic precipitation will not rapidly change pH due to acidic precipitation, but will probably release Al^{3+} ions into the leachate (McFee, 1980). Soils having a low CEC are usually low in plant nutrients; therefore, significant changes in their productivity could occur with only a slight loss of nutrients (McFee, 1980).

Even though CEC or buffering capacity does not completely define soil sensitivity to possible influents of acid, for the reasons given above it was the primary criterion used by McFee for the regional mapping of soil sensitivity to acidic precipitation in the eastern United States. Further, though it is frequently stated in much of the literature that soils with low CEC or sandy soils having low organic matter are likely to be most susceptible to effects of acidic precipitation, the "low CEC" values are not quantified. To develop a working set of classes, it was necessary to make certain assumptions and "worst case" calculations. Since soils in general are rather resistant to change due to additions of acid, a fairly high addition of acid was assumed and the question asked, "What is the maximum effect that it can have on soil, and how high would the CEC have to be to resist that effect?" (McFee, 1980)

To determine sensitivity of a soil, McFee arbitrarily chose a span of 25 years. It was hypothesized that a significant effect could occur if the maximum influx of acid (100 cm of precipitation at pH 3.7 per annum) during that period equaled 10 to 25 percent of the cation exchange capacity in the top 25 cm of soil. Soils are considered slightly sensitive if the top 25 cm of soil has an average CEC of 6.2 to 15.4 meq/100 g (assuming a bulk density of 1.3 g/cc). If the same influx of acid exceeds 25 percent of the CEC in the top 25 cm, i.e., when the CEC is less than 6.2 meq/100 g, the soils are considered sensitive.

Based on the above concepts, the soils of the eastern United States including effects of cultivation were mapped (Figure 11-30) by McFee. The areas containing most of the soils potentially sensitive to acidic precipitation are in the upper Coastal Plain and Piedmont regions of the southeast, along the Appalachian Highlands, through the east central and northeastern areas, and in the Adirondack Mountains of New York (McFee, 1980). The present limited state of knowledge regarding the effects of acidic precipitation on soils makes a more definitive judgment of the location of areas with the most sensitive soils difficult at the present time.

The capacity of soils to absorb and retain anions also important in determining whether soils will become acidified was not discussed by McFee (1980). The capacity for anion absorption is great in soils rich in hydrated oxides of aluminum (Al) and iron (Fe). Reduced leaching of salt cations is of great significance not only in helping to prevent soil acidification but in geochemical circulation of nutrients, fertilization in agriculture and preventing water pollution (Johnson et al., 1980; Johnson, 1980; Wiklander, 1980). (See Section 11.3.2.1) This parameter, as well as those listed by McFee (1980) should be used in determining the sensitivity of soils to acidification by both wet and dry deposition.

11.5 SUMMARY

Occurrence of acidic precipitation (rain and snow) in many regions of the United States, Canada, and Scandanavia has been implicated in the disappearance or reduction of fish, other animals, and plant life in ponds, lakes, and streams. In addition, acidic precipitation possesses the potential for impoverishing sensitive soils, degrading natural areas, injuring forests, and damaging monuments and buildings made of stone.

Sulfur and nitrogen oxides, emitted through the combustion of fossil fuels are the chief contributors to the acidification of precipitation. The fate of sulfur and nitrogen oxides, as well as other pollutants emitted into the atmosphere, depends on their dispersion, transport, transformation and deposition. Emissions from automobiles occur at ground level, those from electric power generators from smoke stacks 1000 feet or more in height. Transport and transformation of the sulfur and nitrogen oxides are in part associated with the height at which they are emitted. The greater the height, the greater the likelihood of a longer residence time in the atmosphere and a greater opportunity for chemical transformation of the oxides to sulfates, nitrates or other compounds. Ozone and other photochemical oxidants are believed to be involved in the chemical transformations. Because of long range transport, acidic precipitation in a particular state or region can be the result of emissions from sources in states or regions hundreds of miles away rather than local sources. To date the complex nature of the chemical transformation processes has not made the demonstration of a direct cause and effect relationship between emissions of sulfur and nitrogen oxides and the acidity of precipitation possible.

Natural emissions of sulfur and nitrogen compounds are also involved in the formation of acidic precipitation; however, in industrialized regions anthropogenic emissions exceed natural emissions.

Precipitation is conventionally defined as being acidic if its pH is less than 5.6. Currently the acidity of precipitation in the northeastern United States, the region most severely impacted, ranges from pH 3.0 to 5.0. Precipitation episodes with a pH as low as 3.0 have been reported for other regions of the United States. The pH of precipitation can vary from event to event, from season to season and from geographical area to geographical area.

The impact of acidic precipitation on aquatic and terristrial ecosystems is not the result of a single or several precipitation events, but the result of continued additions of acids or acidifying substances over time. Wet deposition of acidic substances on freshwater lakes, streams, and natural land areas is only part of the problem. Acidic substances exist in gases, aerosols, and particulate matter transferred into the lakes, streams, and land areas by dry deposition. Therefore all the observed biological effects should not be attributed to acidic precipitation alone.

Sensitivity of a lake to acidification depends on the acidity of both wet and dry deposition, the soil system of the drainage basin, canopy effects of ground cover and the composition of the watershed bedrock.

Ecosystems can respond to environmental changes or perturbations only through the response of the populations of organisms of which they are composed. Species of organisms sensitive to environmental changes are removed. Therefore, the capacity of an ecosystem to maintain internal stability is determined by the ability of individual organisms to adjust their physiology or behavior. The success with which an organism copes with environmental changes is determined by its ability to yield reproducing offspring. The size and success of a population depends upon the collective ability of organisms to reproduce and maintain their numbers in a particular environment. Those organisms that adjust best contribute most to future generations because they have the greatest number of progeny in the population.

The capacity of organisms to withstand injury from weather extremes, pesticides, acidic deposition or polluted air follows the principle of limiting factors. According to this principle, for each physical factor in the environment there exists for each organism a minimum and a maximum limit beyond which no members of a particular species can survive. Either too much or too little of a factor such as heat, light, water, or minerals (even though they are necessary for life) can jeopardize the survival of an individual and in extreme cases a species. When one limiting factor is removed another takes its place. The range of tolerance of an organism may be broad for one factor, narrow for another. The tolerance limit for each species is determined by its genetic makeup and varies from species to species for the same reason. The range of tolerance also varies depending on the age, stage of growth or growth form of an organism. Limiting factors are, therefore, factors which, when scarce or overabundant, limit the growth, reproduction and/or distribution of an organism. The increasing acidity of water in lakes and streams appears to be such a factor. Significant changes that have occurred in aquatic ecosystems with increasing acidity include the following:

1. Fish populations are reduced or eliminated.
2. Bacterial decomposition is reduced and fungi may dominate saprotrophic communities. Organic debris accumulates rapidly, tying up nutrients, and limiting nutrient mineralization and cycling.
3. Species diversity and total numbers of species of aquatic plants and animals are reduced. Acid-tolerant species dominate.
4. Phytoplankton productivity may be reduced due to changes in nutrient cycling and nutrient limitations.
5. Biomass and total productivity of benthic macrophytes and algae may increase due partially to increased lake transparency.
6. Numbers and biomass of herbivorous invertebrates decline. Tolerant invertebrate species, e.g., air-breathing bugs (water-boatmen, back-swimmers, water striders) may become abundant primarily due to reduced fish predation.
7. Changes in community structure occur at all trophic levels.

Studies indicate that pH concentrations between 6.0 and 5.0 inhibit reproduction of many species of aquatic organisms. Fish populations become seriously affected at a pH lower than 5.0.

Disappearance of fish from lakes and streams follows two general patterns. One results from sudden short-term shifts in pH, the other arises from a long-term decrease in the pH of the water. A major injection of acids and other soluble substances occurs when polluted snow melts during warm periods in winter or early spring. Fish kills are a dramatic consequence of such episodic injections.

Long-term increases in acidity interfere with reproduction and spawning, producing a decrease in population density and a shift in size and age of the population to one consisting primarily of larger and older fish. Effects on yield often are not recognizable until the population is close to extinction; this is particularly true for late-maturing species with long lives. Even relatively small increases (5 to 50 percent) in mortality of fish eggs and fry can decrease yield and bring about extinction.

Aluminum is mobilized at low pH values. Concentrations of aluminum may be as or more important than pH levels as factors leading to declining fish populations in acidified lakes. Certain aluminum compounds in the water upset the osmoregulatory function of the blood in fish. Aluminum toxicity to aquatic biota other than fish has not been assessed.

An indirect effect of acidification potentially of concern to human health is possible heavy metal contamination of edible fish and of water supplies. Studies in Canada and Sweden reveal high mercury concentrations in fish from acidified regions. Lead and copper have been found in plumbing systems with acidified water, and persons drinking the water could suffer from lead or copper poisoning.

Acidic precipitation may indirectly influence terrestrial plant productivity by altering the supply and availability of soil nutrients. Adidification increases leaching of plant nutrients (such as calcium, magnesium, potassium, iron, and manganese) and increases the rate of weathering of most minerals. It also makes phosphorous less available to plants. Acidification also decreases the rate of many soil microbiological processes such as nitrogen fixation by Rhizobium bacteria on legumes and by the free-living Azotobacter, mineralization of nitrogen from forest litter, nitrification of ammonium compounds, and overall decay rates of forest floor litter.

At present there are no documented observations or measurements of changes in natural terrestrial ecosystems that can be directly attributed to acidic precipitation. This does not necessarily indicate that none are occurring. The information available on vegetational effects is an accumulation of the results of a wide variety of controlled research approaches largely in the laboratory, using in most instances some form of "simulated" acidic rain, frequently dilute sulfuric acid. The simulated "acid rains" have deposited hydrogen (H^+), sulfate ($SO_4^=$) and nitrate (NO_3^-) ions on vegetation and have caused necrotic lesions in a wide variety of plants species under greenhouse and laboratory conditions. Such results must be interpreted with caution, however, because the growth and morphology of leaves under greenhouse conditions are often atypical of field conditions. Based on laboratory studies, sensitivity of plants to acidic depositions seems to be associated with the wettability of leaf surfaces. The shorter the time of contact, the lower the resulting dose, and the less likelihood of injury.

Erosion of monuments and buildings made of stone and corrosion of metals can result from acidic precipitation. Because sulfur compounds are a dominant component of acidic precipitation and are deposited during dry deposition also, the effects resulting from the two processes cannot be distinguished. In addition, the deposition of sulfur compounds on stone surfaces provides a medium for microbial growth that can result in deterioration.

CHAPTER 11

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12. EFFECTS OF NITROGEN OXIDES ON NATURAL ECOSYSTEMS, VEGETATION AND MICROORGANISMS

12.1 INTRODUCTION

Ecosystems are complex self-sustaining natural systems and are composed of living organisms and the several nonliving components of the environment within which the organisms exist. Within an ecosystem are included all of the interactions that bind the living and nonliving components together into a stable system, i.e., the interactions between organisms or communities of organisms, the relationships between organisms and the physical environment and the interactions of the various aspects of the nonliving environment (Boughey, 1971; Odum, 1971; Smith, 1974; Whittaker, 1975). After centuries of relatively stable annual climatic and geochemical conditions, they may become self-perpetuating (Boughey, 1971; Odum, 1971; Smith, 1974; Whittaker, 1975).

Evaluating the contribution of functioning natural ecosystems to human welfare is a very complex task and involves weighing both economic and human social values; however, it is clear that this natural order by which living organisms are bound to each other and to their environment is essential to the existence of any species on earth, including man (Boughey, 1971; Odum, 1971; Smith, 1974; Whittaker, 1975). As life support systems, the value of ecosystems cannot be quantified in economic terms.

This chapter discusses the effects in general of nitrogen oxides on natural ecosystems and, more specifically, their effects on certain species of plants and microorganisms.

12.2 EFFECTS OF NITROGEN COMPOUNDS ON NATURAL ECOSYSTEMS

Climatic, physicochemical, or biological changes, regardless of their source or nature, will affect the functioning of an ecosystem. Some ecosystems are durable and relatively stable when subjected to a given environmental change; others become unstable given the same change.

It is difficult to assess the complex cause and effect relationships of any pollutant, even when it is studied using only a single organism. When attempting to assess such relationships within populations, communities and ecosystems, the problems become even greater. Additional complications in determining the effects of a single pollutant on natural communities are created by the presence of multiple contaminants that may promote synergistic or antagonistic effects.

The response of ecosystems to environmental changes or perturbations is determined by the response of their constituent organisms. The factors which determine the response of organisms and changes which may occur within ecosystems are discussed in Section 11.1.2 Ecosystem Dynamics in Chapter 11.

Terrestrial, marine, and freshwater ecosystems are functionally important to the integrity of the biosphere. They are important: (1) in the production of food; (2) the maintenance of forests; (3) as global support systems for the regeneration of essential nutrients and atmospheric components; (4) for their aesthetic value in maintaining natural vegetative communities; and (5) in the assimilation or destruction of many pollutants from the air, water, and soil.

Of the many functions occurring within ecosystems, the flow of energy and nutrients are among the most important. Energy flows through an ecosystem in only one direction while nutrients are recirculated (Boughey, 1971; Odum, 1971; Smith, 1974; Whittaker, 1975).

Nitrogen, one of the nutrients recirculated by ecosystems, is an element essential to all life. It is necessary in the formation of the cells of which all living matter is composed. The production of virtually all food depends directly or indirectly on biologically available nitrogen. The most abundant source of nitrogen is the atmosphere of which molecular nitrogen composes 78 percent (Smith, 1974, Whittaker, 1975). However, most organisms are unable to use molecular nitrogen, therefore, it must be converted into another chemical form. Nitrogen is made available to organisms through the conversion or fixation of molecular nitrogen into biologically available compounds. These transformations of nitrogen are regulated almost entirely by terrestrial and aquatic microorganisms through a complicated series of reactions that are collectively termed the "nitrogen cycle." These transformations are more accurately described as a "nitrogen web" because the image of a simple loop of compounds through which nitrogen successively passes bears little resemblance to reality.

The nitrogen cycle or web has been greatly modified by man, both locally and on a global scale, through agricultural activity, industrial production, fuel burning and waste disposal.

The nitrogen cycle in the biosphere and the modifications caused by man's activities are discussed in Chapter 4.

In terrestrial and aquatic systems, the major nonbiological processes of the nitrogen cycle involve phase transformations rather than chemical reactions. These transport processes include volatilization of ammonia and other gaseous forms of nitrogen; sedimentation of particulate forms of organic nitrogen; and sorption (e.g., of ammonium ion by clays). Understanding of the biospheric nitrogen cycle and of the factors that control the cycle depends primarily on an understanding of biological principles, especially those of microbial ecology.

The biological transformations shown in Figure 4-1 (Chapter 4) involve only six major processes (Figure 12-1). These processes are discussed here in outline only. For details see Chapter 4.

These processes are:

1. Assimilation of inorganic forms of nitrogen (primarily ammonia or nitrates) by plants and microorganisms to form organic nitrogen, e.g., amino acids, proteins, purines, pyrimidines, and nucleic acids. In this report the term ammonia is used for gaseous NH_3 and collectively for NH_4+ plus NH_3, when there is no need or intention to distinguish between these forms. Ammonium (ion) is used specifically to indicate the cationic form NH_4+.
2. Heterotrophic conversion of organic nitrogen from one organism (food or prey) to another organism (consumer or predator). Nitrogen is bound in plant or animal protein until the organisms die or as in the case of animals certain products are excreted.
3. Ammonification, the decomposition of organic nitrogen to ammonia. (The ammonia may be assimilated by aquatic or terrestrial plants and microorganisms, may be bound by clay particles

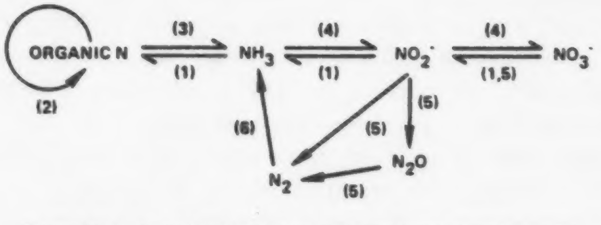
ORGANIC N
(2)
(3)
(1)
NH$_3$
(4)
(1)
NO$_2^-$
(4)
(1,5)
NO$_3^-$
(6)
(5)
(5)
N$_2$O
N$_2$
(5)

Figure 12-1. Simplified biological nitrogen cycle, showing major molecular transformations. Numbers in parentheses correspond to numbered processes discussed in text: (1) assimilation; (2) heterotrophic conversion; (3) ammonification; (4) nitrification; (5) denitrification; (6) nitrogen fixation (National Research Council, 1978).

in the soil, or it may be converted by microorganisms to form nitrates in the process termed nitrification. It may also escape into the air.).

4. Nitrification, the oxidation of ammonium to nitrite and nitrate through microbial action. Nitrates may be assimilated by plants, washed downward through the soil into groundwater or through surface runoff into streams, rivers and oceans or may be transformed into atmospheric nitrogen or reduced to ammonia.

5. Denitrification, implies the gaseous loss of nitrogen, usually as molecular nitrogen (N_2), nitrous oxide (N_2O) or nitric oxide (NO), to the atmosphere as a result of microbial reduction of nitrate. Nitrate is reduced to nitrous oxide (N_2O) and molecular nitrogen (N_2) under anaerobic conditions. Nitrates (NO_3^-) are converted into nitrites (NO_2^-), to nitrous oxide (a gas) (N_2O) and finally into nitrogen gas (N_2) which goes off into the atmosphere. In the soil, nitrites rarely accumulate under acidic conditions but are decomposed spontaneously to nitric oxide (NO), and under alkaline conditions they are biologically converted to N_2O and N_2 (Alexander, 1977; Brock, 1970). It must be emphasized that this process is anaerobic and that conversion of nitrates to nitrites is extremely sensitive to the presence of atmospheric oxygen. If atmospheric oxygen is present, the conversion does not occur. Some evidence exists for the nonbiological chemical production of nitrogen gas or nitrogen oxides (Delwiche and Bryan, 1976).

6. Biological Nitrogen Fixation is the transformation of atmospheric nitrogen gas into ammonia, nitrates and other nitrogen-containing compounds. The transformation is carried out by a variety of microorganisms. The microorganisms may be either symbiotic (living in the roots of leguminous plants) or nonsymbiotic (living independently in the soil) and the process may be accomplished under aerobic or anaerobic conditions.

The predominant agents of assimilation in water are autotrophic algae and on land, higher plants. In soils, bacteria are important agents of assimilation of inorganic nitrogen. Heterotrophic conversions (e.g., of organic nitrogen in plants to animal protein) are highly complicated processes involving numerous steps, but are not treated in any detail here.

Ammonification and nitrification together constitute the process of mineralization. Bacteria and fungi are the principal agents of ammonification in soils; autolysis of cells and excretion of ammonia by zooplankton and fish are important processes in aquatic systems. Ammonification is important in renewing the limited supply of inorganic nitrogen for further assimilation and growth by plants. Nitrification is mediated primarily by aerobic bacteria that obtain their energy by oxidizing ammonia to nitrite and nitrate. Nitrification converts ammonia, which is volatile but readily absorbable, into nitrate, a nonvolatile but easily leached form.

On an ecosystem scale, denitrification is considered a nitrogen sink since the products (N_2 and N_2O) are readily lost to the atmosphere and most organisms cannot use nitrogen in these gaseous forms. Denitrification is carried out by a ubiquitous group of bacteria that use nitrate as their terminal electron acceptor in the absence of oxygen.

Nitrogen fixation is important as a source of available nitrogen for plant growth in both natural and managed agricultural ecosystems. On a global scale and over millions of years, nitrogen fixation balances the losses by denitrification; on time scales of decades to thousands of years, the two processes may be out of phase without significantly affecting the nitrogen content of the global atmosphere.

Nitrogen fixation is only an indirect source of nitrate in the biosphere, but this process is important in global nitrogen balances, and in the current controversy over the depletion of stratospheric ozone by N_2O. (See Chapter 9).

Numerous texts, monographs, and papers review the nitrogen cycle, (Alexander, 1977; Bartholomew and Clark, 1965; Brezonik, 1972, Brock, 1970; Delwiche, 1970; Delwiche, 1976; Delwiche, 1977, Hutchinson, 1944; Hutchinson, 1954; Hutchinson, 1957; Keeney, 1973; Söderlund and Svensson, 1976) and other reviews cover specific aspects of the cycle in detail. This discussion emphasizes the processes of the nitrogen cycle that are important to an understanding of the accumulation of nitrate and its transformations in the biosphere. Because the literature dealing with nitrate and the nitrogen cycle is so extensive, no attempt has been made to provide exhaustive documentation here.

As indicated above, of the many functions occurring within ecosystems, the recycling of nutrients is one of the most important. The nitrogen cycle is one example of the cycling of a very important nutrient. Any effect, environmental or biological, which interferes with the cycling process could be detrimental to the lives of plants and animals.

12.2.1 Effects of Nitrates

Ecological effects of increased nitrates can be beneficial or detrimental or both. Effects of both kinds may occur simultaneously, may cause effects in media or in ecological compartments quite removed from those that initially receive the manmade nitrogenous injections. In some natural ecosystems, such as lakes and estuaries, the addition of nitrogen can contribute to eutrophic conditions that are considered undesirable. Nitrate as nitric acid contributes to the acidity of rainfall (Chapter 11) and some nitrates and related compounds are toxic to plants, animals and microorganisms.

In most nonagricultural terrestrial ecosystems, the major processes that provide nitrogen for plant growth are mineralization (ammonification and nitrification) of soil organic nitrogen and biological fixation of atmospheric nitrogen. When fluxes of nitrogen enter such systems as a result of human activities, the added inputs in many cases represent a significant fraction of total nitrogen inputs. On the basis of such mass-balance considerations, it seems likely that such fluxes are important nutrient sources that could support increased biotic productivity (National Research Council, 1978).

Except in ecosystems that receive fertilizer or nitrogenous wastes, the most important manmade contributions are likely to be from atmospheric pathways, total (inorganic plus organic) nitrogen loadings in wet and dry precipitation may be equivalent to from 8 to 25 percent of the nitrogen used by plants in different natural ecosystems. Even in heavily managed ecosystems, annual atmospheric fluxes may be substantial; for instance, the calculated total nitrogen contribution from precipitation and from gaseous deposition over the Florida peninsula exceed by a factor of two the amount of nitrogen applied as fertilizer to the agricultural land area within the region (National Research Council, 1978).

Predicting the effects of nitrates and other anthropogenic nitrogen compounds on natural ecosystems involves much greater uncertainties than does prediction of the yield response of an agricultural crop. First, it is far more difficult to determine accurately the actual anthropogenic nitrogen contributions to most ecosystems; this is especially true for terrestrial systems, where the major influxes are from atmospheric deposition. Second, far less is known of the responses of nonagricultural plant communities to increased supplies of fixed nitrogen than is known for cultivated crops. It is possible, however, to estimate the approximate magnitude of anthropogenic nitrogen fluxes to ecosystems, using the limited amount of monitoring data available or mass-balance calculations (see Appendix A of the National Academy of Sciences: Nitrates; an Environmental Assessment) (National Research Council, 1978). Such estimates, and quantitative information about the nitrogen cycle at specific sites in the system under study, make it possible to reach some conclusions about the possible ecological significance of the added nitrogen. In addition, where the data base is more extensive, as it is for a number of lakes in various stages of eutrophication, more quantitative dose-response relationships can be estimated.

Living organisms are exposed to nitrates and related compounds through air, water, soil, and food webs. The rate of exposure depends on proximity to sources and on a great many environmental transport and transformation processes (illustrated in Figure 12-2). Biological productivity can be increased deliberately by fertilization, as in agricultural crops, or accidentally through run off to terrestrial or lake systems.

12.2.2 Terrestrial Ecosystems

Additions of nitrate and other forms of nitrogen from the atmosphere to ecosystems is an integral function of the terrestrial nitrogen cycle. Higher plants and microorganisms can assimilate the inorganic forms rapidly. The contribution of inorganic nitrogen in wet precipitation (rain plus snow) is usually equivalent to only a few percent of the total nitrogen assimilated annually by plants in terrestrial ecosystems; however total nitrogen contributions, including organic nitrogen, in bulk precipitation (rainfall plus dry fallout) can be significant, especially in unfertilized natural systems.

In absolute terms, atmospheric contributions of nitrate can range from less than 0.1 kg N/ha-yr in the Northwest (e.g., Fredericksen, 1972) to 4.9 kg N/ha-yr in the eastern United States (Henderson and Harris, 1975; Likens et al., 1970). Inorganic nitrogen (ammonia-N plus nitrate-N) loadings in wet precipitation ranged from less than 0.5 kg/ha-yr to more than 3.5 kg/ha-yr in Junge's (1958) study of rainfall over the United States. On the other hand, total nitrogen loads in bulk precipitation range from less than 5 kg/ha-yr in desert regions of the West to more than 30 kg/ha-yr near barnyards in the Midwest. Total contributions of nitrogen from the atmosphere commonly range from about 10 to 20 kg N/ha-yr for most of the United States (National Research Council, 1978).

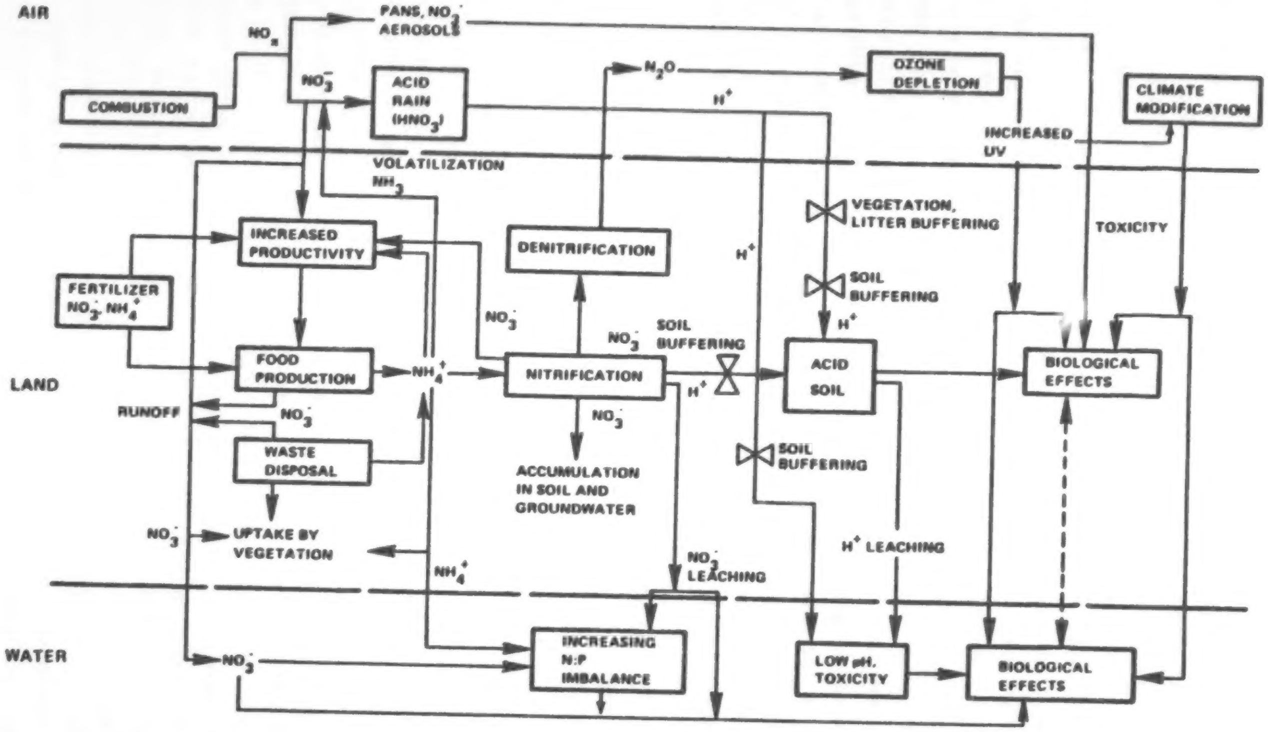
AIR
LAND
WATER
COMBUSTION
NO$_x$
PANS, NO$_3^-$ AEROSOLS
NO$_3^-$
ACID RAIN (HNO$_3$)
VOLATILIZATION NH$_3$
N$_2$O
H$^+$
OZONE DEPLETION
INCREASED UV
CLIMATE MODIFICATION
TOXICITY
FERTILIZER NO$_3^-$, NH$_4^+$
INCREASED PRODUCTIVITY
FOOD PRODUCTION
NH$_4^+$
DENITRIFICATION
NITRIFICATION
NO$_3^-$
SOIL BUFFERING
VEGETATION, LITTER BUFFERING
ACID SOIL
BIOLOGICAL EFFECTS
RUNOFF
WASTE DISPOSAL
UPTAKE BY VEGETATION
ACCUMULATION IN SOIL AND GROUNDWATER
NO$_3^-$ LEACHING
H$^+$ LEACHING
INCREASING N:P IMBALANCE
LOW pH, TOXICITY
BIOLOGICAL EFFECTS

Figure 12-2. Schematic presentation of environmental effects of manipulation of the nitrogen cycle. Human-caused perturbations are shown at left, culminating in ecological and climatic effects, at right. Processes that buffer against the effects are indicated with the symbol(⋈) on the arrows representing the appropriate pathways (National Research Council, 1978);

In comparison, rates of annual uptake by plants range from 11 to 125 kg N/ha-yr in selected ecosystems from several bioclimatic zones (National Research Council, 1978). Since the lowest additions are generally associated with desert areas, where rates of uptake by plants are low, and the highest additions usually occur in moist areas where plant uptake is high, the contributions of ammonia and nitrate from rainfall to terrestrial ecosystems are equivalent to about 1 to 10 percent of annual plant uptake. The typical fluxes (additions) of total nitrogen in bulk precipitation, on the other hand, represent from about 8 to 25 percent of the annual plant needs in eastern deciduous and western coniferous forest ecosystems. Although these comparisons suggest that plant growth in terrestrial ecosystems depends to a significant extent on atmospheric loadings, it is not yet possible to estimate the importance of these contributions when compared to biological nitrogen fixation and mineralization of nitrogen in the soil. In nutrient-impoverished ecosystems, such as badly eroded abandoned croplands or soils subjected to prolonged leaching by acid precipitation, nitrogen additions from atmospheric fluxes are certainly important to biological productivity. Such sites, however, are relatively limited in extent. In largely unperturbed forests, recycled nitrogen from the soil organic pool is the chief source of nitrogen for plants, but new nitrogen to support increased production must come either from biological fixation or from atmospheric influxes. It seems possible, therefore, that man generated contributions could play a significant ecological role in a relatively large portion of the forested areas near industrialized regions.

12.2.3 Effects of Nitrogen Oxides

12.2.3.1 Terrestrial Plant Communities--Studies of plant communities suggest that individual species differ appreciably in their sensitivity to chemical stress, and that such differences are reflected in the changes occurring within plant communities. A common alteration in a community under stress is the elimination of the more sensitive populations and an increasing abundance of species that tolerate or are favored by the stress (Woodwell, 1970). The response of plant populations or species to an environmental perturbation will depend upon life cycles of the plants, microhabitats in which they are growing, and their genetic constitution (genotype). Abundant evidence exists to show that in plant communities undergoing structural changes that reduce environmental or biological variability new species become dominant (Botkin, 1976; Daniel, 1963; Jordan, 1969,; Keever, 1953; McCormick, 1963; McCormick, 1969; Miller, 1973; Miller and Yoshiyama, 1973; Smith, 1974; Treshow, 1968; Woodwell, 1962; Woodwell, 1963; Woodwell, 1970). Furthermore, the specific pollutant to which a community is exposed for prolonged periods of time will govern the capacity of the community to recover. In turn, an alteration in the community composition, size of the community, or its rate of energy fixation will influence the animal populations in the vicinity and microorganisms in the underlying soil. These changes, in their turn, will modify the behavioral patterns or alter competition among the prevalent organisms.

Investigations of the influence of nitrogen oxides on economically important plant species have revealed differential susceptibility of plant species, differential effects due to diurnal conditions and age, and difficulties in predicting synergistic effects when a plant population is exposed to more than one pollutant simultaneously. Little information is known about the specific effect of nitrogen oxides on plants in natural communities. Evidence of visible injury to plant communities has seldom been demonstrated. In fact, visible injury may represent only a fraction of the actual harm done to terrestrial communities. Evidence exists, however, that the vigor and survival rate of plants have been affected deleteriously by air pollution (Hepting, 1964). Many instances of injury to higher plants that have been ascribed to pathogens or unknown factors may, in fact, reflect a toxicity associated with nitrogen oxides. Nevertheless, since plants respond simultaneously to many environmental factors, it is frequently difficult to determine which of the potential environmental stresses are responsible for damage to the major species or to the community composition. Thus, it is likely that the response to modest nitrogen oxide stresses would not be recognized because the vigor and visual appearance of plants would be influenced by temperature, soil type and moisture, drainage, interspecific competition, and other factors. Only severe injury could likely be ascribed to a particular pollutional episode (Hepting, 1964).

The agent of stress may have ecological importance inasmuch as the species tolerance to such environmental factors as moisture, temperature, and light, its capacity to compete, and its ability to withstand attack by parasitic organisms, may be affected as well. Moreover, physiological aspects of plant development, including growth, photosynthetic and respiratory rate, and flowering may be influenced by the pollutant. These alterations in the environment and in the plant community will influence energy flow through the ecosystem, its productivity, and the succession of indigenous species.

Studies have been conducted on the effect of air pollutants on ecosystems and plant communities (Parmeter and Cobb, 1972; Wenger et al., 1971). It is the general conclusion of these investigations that further research on the influence of nitrogen oxides on plant communities is required. The available information clearly is too small to warrant meaningful generalizations at this time; however, there is information detailing the effects on individual plant species. These effects are discussed in Section 12.3.

12.2.3.2 Effects on Animal Communities--Surprisingly little attention has been given to the effect of nitrogen oxides on animal populations or communities. Although laboratory studies of a few individual species have been carried out, it is difficult to extrapolate from these laboratory tests on animals maintained under careful conditions, to populations in the field. The interaction of the various stresses in nature and the uncertainty of cause and effect relationships make any conclusions from laboratory studies quite tenuous. Because species differ enormously in their susceptibility to air pollutants, extrapolation from laboratory tests on one species to potential effects on another is fraught with problems.

One of the few studies conducted on animal populations is that of McArn et al. (1974) who reported that granule-rich microphages appeared in the lung tissues of English sparrows nesting in urban areas with high pollution levels. The microphages were not reported to be present in the lungs of sparrows inhabiting windswept, unpolluted areas. In this study, potential chronic effects could not be determined owing to the relatively short life-span of these birds.

Beyond this limited amount of knowledge, the literature concerning the effects of nitrogen oxides on natural animal populations or communities is extremely sparse. No conclusions can be drawn about whether ambient levels of nitrogen oxides in the atmosphere have an effect on the composition or functioning of animal communities or populations.

12.2.3.3 Effects of Nitrogen Oxides on Microbial Processes in Nature--Microorganisms are essential for the functioning of key processes in terrestrial, marine, and freshwater communities. They are the chief agents for decomposing organic materials in soils and waters. Microfloras are the major agents for destruction of synthetic chemicals introduced into soils and waters. Marine algae are essential for the generation of the oxygen required to sustain life in all higher animals. In soil, the bacteria, fungi, and actinomycetes convert compounds of nitrogen, sulfur, and phosphorus to the inorganic state, thereby providing plants with the required inorganic nutrients. Biologic nitrogen fixation and nitrification are affected solely by these microscopic organisms, which also maintain soil structure and form the humus important to abundant plant growth. In addition, many of the pathogens that are constantly discharged into soils and waterways are eliminated by microbial actions.

Since microorganisms are critical to the balance of ecosystems, any disturbance in their activities could have serious consequences on a local, regional, or global scale. The potential impact on microorganisms by substances as widespread and pervasive as the nitrogen oxides must therefore be assessed. Surprisingly, this subject has been neglected to date. The few data are based on NO_x concentrations in excess of those found in the atmosphere.

Therefore, the knowledge concerning the potential impact of nitrogen oxides on microbial processes in soils and waters is sparse. Although ambient concentrations probably do not significantly affect biologic processes in natural ecosystems, it is not possible to support this view with experimental data (National Academy of Sciences, 1977).

12.2.4 Aquatic Ecosystems, Nitrogen and Eutrophication

The overenrichment of surface waters, usually lakes, with nutrients is termed eutrophication. This process results in an array of water quality changes that are generally regarded as undesirable. Phosphorus and nitrogen are the most important nutrients that stimulate eutrophication, and in most lakes phosphorus is considered the more critical of the two. In coastal and estuarine ecosystems, however, nitrogen is more often the limiting nutrient and nitrogen inputs may control eutrophication. Furthermore, in many already-eutrophic lakes, biotic productivity is controlled by nitrogen, because the N/P ratios of pollutants from many cultural sources (e.g., domestic sewage) are far below the ratios needed for plant growth. The role of nitrogen in cultural eutrophication therefore appears to be important, although it is complex and poorly quantified relative to the role of phosphorus.

The sources of manmade nitrogen reaching surface waters include sewage, industrial wastes, animal manures, surface runoff and sub-surface transport of nutrients from urban and agricultural lands, and atmospheric fluxes. It has been estimated (National Research Council, 1978) that more than 90 percent of the nitrogen entering surface waters comes from nonpoint sources, and that more than 80 percent of that portion is from agricultural lands (including livestock feedlots). Because nitrogen forms in aquatic systems are readily interconvertible, all nitrogen inputs, rather than nitrate per se, must be considered.

The average atmospheric input of 10 to 20 kg N/ha-yr that is typical for most of the United States is also a sufficient nutrient loading to support a moderate increase in biotic productivity in some lakes, especially shallow, oligotrophic lakes that may be nitrogen-limited. Atmospheric nitrogen fluxes may contribute to slight eutrophication in such cases; however, it is unlikely that these inputs alone would induce serious water quality problems.

12.2.4.1 Eutrophication of Lakes--Cultural or man-induced eutrophication has been one of the most intensively studied water quality problems in the past ten to fifteen years. Although many lakes become naturally more productive and nutrient-rich as they fill in and age, natural eutrophication is a slow process, and its effects are unlikely to be perceived within a single human lifetime. However, addition of excessive amounts of nutrients from sewage effluents, agricultural runoff, urban runoff and other anthropogenic sources can greatly modify the characteristics of a lake in a matter of a few years; the literature is replete with examples of this phenomenon.

The over-enrichment or eutrophication of surface waters, usually lakes, with nutrients results in an array of water quality changes that are generally considered undesirable. These changes most commonly include the proliferation or "blooms" of algae and aquatic macrophytes, the depletion of dissolved oxygen in bottom water, a decrease in water clarity, the loss of cold water fisheries, shortened food chains, and takeover by rough fish. Table 12-1 summarizes changes in common trophic state indicators that occur when lakes become eutrophic, and Table 12-2 lists some common water use problems that may result from eutrophication.

Eutrophication is usually considered undesirable. This somewhat narrow viewpoint, however, ignores the fact that nutrient-rich waters are more productive not only of algae, but also of fish. Lakes are not now a significant source of protein in the United States, but lake fish may be an important food resource in a food-hungry world. Many sports fishermen prefer moderately eutrophic lakes, unless they are seeking coldwater fish, which cannot survive in such lakes because of oxygen depletion in the cold bottom waters. A conflict thus exists between the desires of some fishermen and the preferences of swimmers and other recreational users of lakes, who generally favor the clearest and most oligotrophic situation. On the other hand, continued nutrient enrichment eventually is undesirable to sport fishermen also, since game fish disappear, rough fish predominate, and excessive aquatic weed growths may hinder or prevent boating in highly eutrophic lakes.

For some functions of some oligotrophic lakes, where nitrogen may be the limiting nutrient, the contribution from runoff or atmosphere fluxes may be essential to maintaining biological productivity. The point at which the effects on productivity of nitrate input to

TABLE 12-1. COMMON TROPHIC STATE INDICATORS AND THEIR RESPONSES TO EUTROPHICATION

Physical Indicators

Transparency (D)[1]
  (Secchi disc reading)

Chemical Indicators

Nutrient concentrations (I)[1]
  (e.g., annual average and spring maximum)
Conductivity (I)
Dissolved solids (I)
Hypolimnetic oxygen (D)
  (generally goes to zero except in very deep eutrophic lakes)
Epilimnetic oxygen supersaturation (I)

Biological Indicators[2]

Algal bloom frequency (I)
Algal species diversity (D)
Chlorophyll *a* (I)
Proportion of blue green algae in plankton (I)
Primary production (I)
Littoral vegetation (I)
Zooplankton (I)
Fish (I)
Bottom fauna (I)
Bottom fauna diversity (D)

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[1] (I) after parameter signifies that value increases with eutrophication; (D) signifies that value decreases with eutrophication.

[2] Biological parameters have important qualitative changes, i.e., species changes as well as quantitative (biomass) changes as eutrophication proceeds.

SOURCE: (Brezonick, 1969).

TABLE 12-2. WATER USE PROBLEMS RESULTING FROM EUTROPHICATION

Water Treatment Problems

- Increased color and turbidity in raw water
- Increased taste and odor (necessitating the use of activated carbon)
- Increased chlorine demand
- Shortened filter runs

Recreational Problems

- Loss of desirable fish and increase in rough fish
- Increased costs in boat and dock maintenance resulting from fouling
- Boat access problems from aquatic vegetation
- Economic losses to owners of resorts and fish camps as fewer people swim, fish and boat in lakes with algal blooms
- Public health problems--swimmers' diseases (mainly eye, ear, nose and throat infections)
- General loss in lake's aesthetic appeal

Agricultural Problems

- Transmissibility of water in canals impaired by extensive macrophyte growths
- Toxicity of algal blooms to cattle and wildlife
- Increases in water loss in arid regions caused by evapotranspiration from floating vegetation

SOURCE: (Brezonick, 1969).

aquatic ecosystems cease to be beneficial is influenced by a number of factors. Some of these are discussed below.

Because phosphorus and nitrogen are the nutrients that limit production in most lakes, these two nutrients are most important in stimulating eutrophication (Vollenweider, 1968). Oligotrophic lakes (low in nutrients) are commonly thought to be phosphorus-limited (Deevey, 1972; Hutchinson, 1973), because of the relative paucity of phosphorus in the biosphere compared to nitrogen, and because the phosphorus in minerals and soils is relatively immobile, whereas nitrogen compounds are quite mobile. Lake Tahoe (California, Nevada) is a well-known example of a nitrogen-limited oligotrophic lake. In highly eutrophic lakes nitrogen is frequently the limiting nutrient, most often because domestic sewage, the chief nutrient source for many eutrophic lakes, is imbalanced with respect to nitrogen and phosphorus. The total N/P ratio (by weight) in sewage is about 3:1 to 4:1, largely because of the widespread use of phosphate detergents. By comparison, the annual N/P ratio of healthy plants is about 7:1 to 8:1 (by weight).

Miller et al. (1974) conducted algal nutrient bioassays on waters from 49 lakes throughout the United States and found that phosphorus limited growth in 35 lakes; nitrogen was limiting in eight lakes; and some other nutrient was limiting in six. The incidence of phosphorus limitation was lower among lakes that were more productive. The same relationship has been noted in National Eutrophication Survey data on Florida lakes (National Research Council, 1978).

12.2.4.2 Eutrophication in Coastal Waters--Studies of estuarine waters at several locations along the east coast of the United States have indicated that low concentrations of dissolved nitrogen limit primary production (Goldman et al., 1973; Goldman, 1976; Ryther and Dunstan, 1971; Thayer, 1971).

Additions of nitrate to such estuarine systems stimulate primary production and can produce changes in the dominant species of plants, leading to cultural eutrophication and ultimately to deterioration of water quality. However, the significance of nitrogen as a limiting nutrient varies in different estuaries and even on a spatial and temporal basis within a single estuarine system (e.g., Thayer (1971), Estabrook (1973), Goldman (1976)). Furthermore, not all estuaries are nitrogen-limited; Myers (1977) found that phosphate was the primary limiting nutrient in near-shore waters of the Gulf of Mexico near Appalachicola, Florida. The high degree of heterogeneity in the role of nitrogen as a control of productivity in coastal areas makes it difficult to establish quantitative relationships between nitrate loading and water quality.

The reasons that nitrogen is more important as a limiting nutrient in marine coastal waters than in fresh waters are uncertain. A higher rate of phosphorus exchange between sediment and water in saline waters is one possibility. It has also been suggested that denitrification of the nitrate that diffuses into anoxic sediments limits the amount of available nitrogen in estuarine areas, but this hypothesis needs further study.

A number of symposia have treated the causes and consequences of eutrophication in considerable detail (Allen and Kramer, 1971; Likens, 1971; Middlebrooks et al., 1973; National

Research Council, 1969). The problem of cultural eutrophication clearly is not solely a nitrogen-related phenomenon, nor is nitrate the only or often even the main form of nitrogen input. Our focus in this section is on current efforts to quantify the relationships between nutrient loading and trophic states, and on evidence for the extent to which nitrate contributes to eutrophication problems.

Sawyer (1947) was the first to determine critical nutrient levels associated with water quality degradation in lakes. He concluded from a study of 17 lakes in southeastern Wisconsin that lakes with spring maximum concentrations of more than 300 mg/l of inorganic nitrogen and more than 10 to 15 mg/l of orthophosphate-P could be expected to have algal nuisances in the summer. These numbers have been widely quoted and used as water quality guidelines in many areas of the United States, in spite of the narrow data base from which they were developed.

Vollenweider (1968), in a classic study, developed the concept of nutrient loading rates and presented graphs of critical areas nutrient loading (grams of nitrogen or phosphorus per square meter of lake surface per year) versus mean depth, as management guidelines. Figure 12-3 presents Vollenweiger's loading graph for nitrogen. Vollenweider (1968) presented a semi-theoretical mass balance nutrient model as the basis for his loading graphs.

Simple nutrient input-output models have provided insights into the dynamics of nutrients in lakes, and they offer a rational basis for the development of critical nutrient loading rates and lake management guidelines (e.g., Dillon and Rigler,1975). Such models to date, however, have been oriented primarily toward phosphorus, under the assumption that it is the key limiting nutrient in lakes. Further studies are needed to develop more accurate loading guidelines for nitrogen and to obtain quantitative data to apply the input-output models to nitrogen-limited systems.

12.2.4.3 Nitrogen Cycling in Eutrophic Lakes--Eutrophication leads not only to increased rates of nitrogen cycling in lakes; it also provides conditions for some reactions in the nitrogen cycle that normally do not occur in oligotrophic lakes. For example, nitrogen fixation by blue-green algae is essentially limited to eutrophic lakes (Horne, 1977; Stewart et al., 1971). Although blue-green algae are cosmopolitan, they are seldom the dominant phylum in oligotrophic lakes, and nitrogen-fixing species (e.g., *Anabaena* spp., *Aphanizomenon flos-aquae*) are rare in non-eutrophic lakes. This fact is ironic in view of the well-known inhibition of fixation by high concentrations of inorganic nitrogen. However, fixation in eutrophic lakes is generally associated with nitrogen depauperate periods, such as late summer in temperate surface waters. Maximum bloom development by nitrogen-fixing blue-green algae requires an adequate supply of phosphorus, and dissolved phosphorus is usually growth-limiting in oligotrophic waters. For example, Vanderhoef et al. (1974) studied nitrogen fixation in Green Bay (Lake Michigan) and found that the nonfixing blue-green *Microcystis* predominated in areas where all nutrients were high. Nitrogen-fixing *Aphanizomenon* increased with declining combined nitrogen concentrations and showed increased efficiency of fixation as inorganic nitrogen levels decreased. The standing crop of this species decreased with decreasing phosphate concentrations. Finally, diatoms

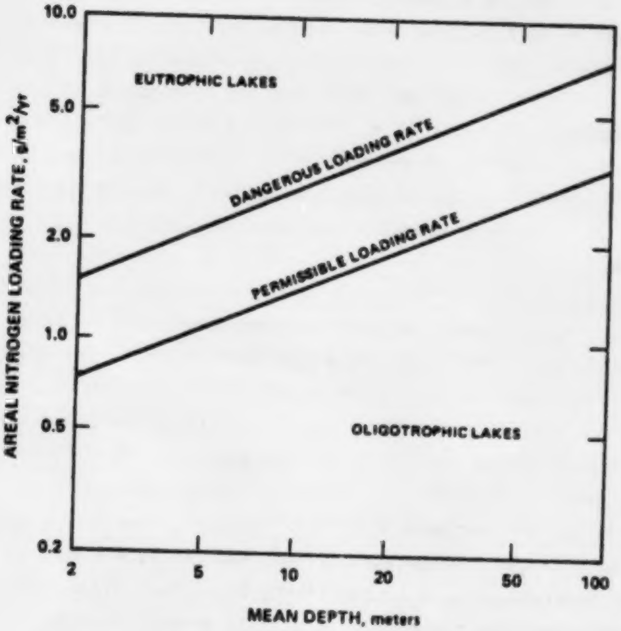

Figure 12-3. Areal loading rates for nitrogen plotted against mean depth of lakes (Vollenweider, 1968).

predominated in the northern reaches of the bay (40 km from the Fox River, the major tributary and source of nutrients for the bay).

The importance of nitrogen fixation in the nitrogen budgets of lakes is controversial. Most reports indicate relatively low contributions (<15 percent) in lakes where fixation occurs at all, but a few cases where fixation supplies up to 50 percent of the annual nitrogen input have been reported. Even in the typical case where fixation makes only a small contribution to the total loading, however, the process is still significant in maintaining nuisance blue-green algal blooms in lake surface waters.

Denitrification occurs in the anoxic hypolimnia of stratified eutrophic lakes, and can represent a significant term in lacustrine nitrogen balances (see National Research Council, Nitrates, 1978). Denitrification also occurs in the anoxic sediments of lakes. The source of nitrate for sediment denitrification may be upward seepage of groundwater, downward diffusion of nitrate from the lake water column, or nitrification in the oxygenated surface layer of sediment. Sediment denitrification can occur in oligotrophic lakes, since their sediments are also anoxic. However, Chen et al. (1972) reported much higher rates in sediments from a hard water eutrophic lake than in those from a soft water oligotrophic lake.

12.2.4.4 Form of Nitrogen Entering Lakes--It is difficult to generalize about the percentage of the total nitrogen loading to lakes that is contributed as nitrate. Nutrient budgets are generally presented by source (streams, rainfall, sewage effluents, etc.) rather than by nitrogen form. Lake Wingra, Wisconsin (National Research Council, 1968) represents one of the few cases where nitrogen loading rates have been broken down according to form. Table 12-3 indicates that 47 percent of the total nitrogen loading to Lake Wingra was in the form of nitrate.

TABLE 12-3. NITRATE-N LOADINGS TO LAKE WINGRA

Source	Kilograms NO_3 -N/yr	Percent Total N as NO_3 -N
Precipitation on lake surface	440	40
Dry fallout	480	22
Spring flow	4,140	96
Urban runoff	600	13
Average	---	--
TOTAL	5,660	47

SOURCE: (National Research Council, 1978).

Inorganic nitrogen forms in lake water are so readily interconvertible that there is probably little to be gained from detailed analysis of this topic. Measured concentrations of ammonia and nitrate in rainfall are roughly comparable, although large short-term, local, and regional variations occur (National Research Council, 1978). Rainfall in industrialized and urbanized regions has exhibited increasing nitrate levels over the past several decades (Likens, 1972). Urban runoff and sewage effluents vary widely in their nitrogen compositon, making generalizations tenuous. Kluesener and Lee (Kluesener, 1972; Kluesener and Lee, 1974) summarized average nitrogen component concentrations from several urban runoff studies. The grand means of the data they collected are: NH_3-N, 0.44 mg/liter; NO_3^--N, 0.51 mg/liter; organic nitrogen, 2.0 mg/liter.

In summary, the contribution of nitrate to eutrophication is uncertain, because of a lack of data dealing with nitrate per se, and due to the ease with which various forms of nitrogen are interconverted. Nitrogen contributions from human activities can promote increased biological productivity in aquatic systems, but the role of nitrogen in eutrophication is understood much less quantitatively than the role of phosphorus. The effects that nitrogen additions may have on productivity, phytoplankton succession, and other processes within aquatic ecosystems are certain to be influenced by other variables, such as light and temperature; however, there is little quantitative information available regarding the relationships among these factors.

12.2.5 The Value of a Natural Ecosystem

Ecosystems are usually evaluated by modern man solely on the basis of their economic value to him, i.e., dollars and cents value to man. This economic value, in turn, is dependent on the extent to which man can manipulate the ecosystem for his own purpose. This single-purpose point of view makes it difficult to explain the many benefits of a natural ecosystem to man's welfare in terms of the conventional cost-benefit analysis. Natural forests are among the most efficient in the fixation of solar energy. Most agriculture, by comparison, is inefficient in total energy fixed; however, in transforming solar energy into food for man it may be highly efficient, so agriculture is emphasized (Woodwell, 1978).

Many functions of natural ecosystems and their benefits to man are unknown to the decision makers. Gosselink, Odum, and Pope (1975) have, however, placed a value on a tidal marsh by assigning monetary values to the multiple contributions to man's welfare such as fish nurseries, food suppliers, and waste-treatment functions of the marsh. They estimate the total social values to range from $50,000 to $80,000 per acre.

Using four different categories, Gosselink, Odum and Pope (1975) developed a step-wise means of assessing the true value of natural tidal marshes to society as a whole. The value was based on commercial usage, social usage and the monetary value of natural ("undeveloped") estuarine environments.

The categories or levels of marshlands to which monetary values were assigned are: (1) Commercial and recreational use, e.g., shell fish production and sport fishing; (2) potential for development, e.g., aquaculture, draining for industrial use; (3) waste assimilation or treatment, e.g., tertiary sewage treatment, and (4) total life support values, e.g., global cycling of nitrogen and sulfur, as protective "breakwaters." The round-figure values calculated in terms of (a) annual return and (b) an income-capitalized value were: (1) a. $100; b. $2,000; (2) a. $1,000; b. $20,000; (3) a. $2,500; b. $50,000; and (4) a. $4,100; b. $82,000.

The foregoing estimates for category (1) were based on identifiable 1974 commercial and recreational uses for which monetary values could be determined rather well. For category (2) the income-capitalization approach was used to estimate the values for development potential and for aquaculture. The estimates for tertiary sewage treatment (3) and life support (4) represent estimates of what man would have to pay for this useful work that is now performed by an acre of estuary were it not available to do this work.

Shortcomings exist in evaluating the environment solely in terms of direct uses or products. "Such cost-accounting ignores the extremely valuable life-support work that natural areas carry on without any development or direct use by man. It is this 'free work of nature' that is grossly undervalued, simply because it has always been taken for granted or assumed to be unlimited in capacity" (Gosselink et al., 1975). Development by man of a salt marsh may adversely affect its functioning in tertiary sewage treatment or as a life support system, therefore, it is important to evaluate it before deciding what kind of development, if any, is in the long-term best interest of both the environment and the economy (Gosselink et al., 1975).

Westman (1977) also evaluated the benefits of natural ecosystems by estimating the monetary costs associated with the loss of the free services (absorption of air pollution, production of oxygen, regulation of global climate and radiation balance, and soil binding) provided by the ecosystems. Westman estimated that the oxidant damage to the San Bernardino National Forest could result in a cost of $27 million per year (1973 dollars) for sediment removal alone due to erosion as long as the forest remained in the early stages of succession.

Estimates of the cost in currency of the values of items and qualities such as clean air and water, untamed wildlife, and wilderness, once regarded as priceless, are an attempt to rationalize the activities of civilization (Westman, 1977). When estimating the monetary cost in currency of the values lost through the damaging of ecosystems, the assumption is usually made that the decision makers will choose the alternative which is most socially beneficial as indicated by costs compared to benefits. As Westman (1977) points out, the assumption "that decisions that maximize benefit cost ratios simultaneously optimize social equity and utility" are based on certain inherent corollaries. These are:

> "(1) The human species has the exclusive right to use and manipulate nature for its own purposes. (2) Monetary units are socially acceptable as means to equate the value of natural resources destroyed and those developed. (3) The value of services lost during the interval before the replacement or substitution of the usurped resource has occurred is included in the cost of the damaged resource. (4) The amount of compensation in monetary units accurately reflects the full value of the loss to each loser in the transaction. (5) The value of the item to future generations has been judged and included in an accurate way in the total value. (6) The benefits of development

> accrue to the same sectors of society, and in the same proportions, as the sectors on whom the costs are levied, or acceptable compensation has been transferred. Each of these assumptions, and others not listed, can and have been challenged" (Westman, 1977).

In the case of (4) above, for example, the losses incurred when the development of natural ecosystems are involved, include species other than man. These losses are seldom, if ever, compensated. The public at large also is usually not consulted to determine whether the dollar compensation is adequate and acceptable. Frequently, there is no direct compensation. Corollary (5) can never be fulfilled because it is impossible to determine accurately the value to future generations.

It should be remembered that ecosystems are life support systems and therefore their worth, in the final analysis, cannot be valued in dollars and cents.

12.3 EFFECTS OF NITROGEN OXIDES ON VEGETATION

Of the various nitrogen oxides (NO_x) in the ambient air (Chapter 8) only nitric oxide (NO) and nitrogen dioxide (NO_2) are considered important phytotoxicants. The direct effects of NO_x on vegetation are usually associated with and confined to areas near specific industrial sources. For example, vegetation injury from exposure to NO_2 has been observed near nitric acid factories and arsenals, but there are no published reports on vegetation injury in the field due to NO or other oxides of nitrogen.

The direct effects of NO_x on vegetation are reviewed in this chapter with emphasis on studies relating NO_x effects to known exposure concentrations and durations. Since most available data pertained to NO_2, this pollutant receives the most attention. Also, when NO_2 was experimentally combined with other pollutants such as SO_2, injury occurred at much lower doses than had been found in earlier studies with NO_2 alone. This suggests that, in certain circumstances NO_2 in conjunction with other gases in the ambient air may behave synergistically.

The contribution of NO_x to the increased acidity of precipitation and its effects on ecosystems is discussed in Chapter 11.

12.3.1 Factors Affecting Sensitivity of Vegetation to Oxides of Nitrogen

A notable feature of the response of vegetation to NO_2 stress is the varied degrees of NO_2-induced injury. This variation ranges from overt leaf chlorosis and necrosis to alterations of leaf metabolism. These differing responses can be explained by the physiological processes affecting NO_2 uptake into the leaf, pollutant toxicity at target sites and cellular repair capacity. Since plants develop as a consequence of environmental-genotypic interactions, each plant possesses a unique set of structural and functional properties which change continuously in response to genetic and environmental stimuli. These interactions between environmental and genetic factors underlie and explain the dissimilar plant responses to NO_2 exposures.

Information on relative sensitivity (differential response) to NO_2 is summarized in Table 12-4. The three classes - susceptible, intermediate, and tolerant - are approximate because

TABLE 12-4. RELATIVE SENSITIVITY OF SEVERAL PLANT SPECIES TO NITROGEN DIOXIDE (HECK AND TINGEY, 1979; MACLEAN, 1977; TAYLOR AND MACLEAN, 1970; TAYLOR ET AL., 1975; U.S. ENVIRONMENTAL PROTECTION AGENCY, 1971)

Plant Type	Susceptible	Intermediate	Tolerant
Coniferous Trees	Larix decidua Mill. (European Larch) Larix leptolepis Gord. (Japanese larch)	Abies alba Mill. (White Fir) Abies homolepis Sieb. & Zucc. (Nikko or Japanese fir) Abies pectinata DC (Common Silver Fir) Chamaecyparis Lawsoniana [Murr.] Parl (Lawson's cypress) Picea glauca [Moench] Voss (White Spruce) Picea pungens glauca, Regel (Colorado Blue Spruce)	Pinus Mugo Turra (Knee pine or dwarf mountain pine) Pinus nigra Arnold (Austrian pine) Taxus baccata L. (English yew)
Field Crops & Grasses	Avena sativa L. (Oats) cv. Clintland 64 cv. 329-80 cv. Pendek Bromus inermis, L. (Bromegrass) cv. Sac Smooth Hordeum distichon L. (Barley) Medicago sativa, L. (Alfalfa) Nicotiana glutinosa L. (Tobacco) Nicotiana tabacum L. (Tobacco) Scorzonera hispanica L. (Viper's grass) T. incarnatum L. (Crimson or Italian Clover) Trifolum pratense L. (Red clover) Triticum vulgare, Vill. (Wheat) cv. Wells Vicia sativa L. (Spring vetch)	Gossypium hirsutum, L. (Cotton) cv. Acala 4-42 cv. Paymaster Nicotiana tabacum, L. (Tobacco) cv. White Gold cv. Bel-B cv. Bel W3 Poa annua, L. (Annual bluegrass) Secale cereale L. (Rye) Triticum aestivum L. (Wheat) Zea Mays L. (Sweet Corn)	Nicotiana tabacum, L. (Tobacco) cv. Burley 21 Poa pratensis L. (Kentucky bluegrass) Sorghum sp. (Sorghum) cv. Martin Zea Mays L. (Corn) cv. Pioneer 509-W cv. Golden Cross
Fruit Trees	Malus sp. (Showy apple)	Citrus sp. (Orange, grapefruit, tangelo)	Hosta plantaginea (Lam.) Aschers (Fragrant plantian lily)

TABLE 12-4 (continued)

Plant Type	Susceptible	Intermediate	Tolerant
	Malus sylvestris Mill. (Apple) Pyrus communis L. (Wild Pear)	Citrus sinensis (L.) Osbeck (Navel Orange)	
Garden Crops	Allium porrum L. (Leek) Apium graveolens L. (Celery) Brassica oleracea botrytis, L. (Broccoli) cv. Calabrese Daucus carota L. (Carrot) Lactuca sativa, L. (Lettuce) Petroselinum hortense Hym. (Parsley) Phaseolus vulgaris, L. (Bean) cv. Pinto Pisum sativum L. (Pea) Raphanus sativus L. (Radish) cv. Cherry Belle Rheum rhaponticum L. (Rhubarb) Sinapis alba (White mustard)	Apium graveolens rapaceum (Celery) Cichorium Endivia, L. (Endive) Ruffec Fragaria chiloensis grandiflora (Pine strawberry) Lycopersicon esculentum, Mill (Tomato) cv. Roma Phaseolus vulgaris humilis Alef. (Bush bean) Solanum tuberosum L. (Potato)	Allium cepa L. (Onion) Asparagus officinalis L. (Asparagus) Brassica caulorapa Pasq. (Kohlrabi) Brassica oleracea acephala DC (Kale) Brassica oleracea capitata L. (Cabbage) Brassica oleracea capitata rubra L. (Red cabbage) Cucumis sativus, L. (Cucumber) cv. Long Marketeer Phaseolus vulgaris, L. (Bush Bean)
Ornamental Shrubs and Flowers	Antirrhinum majus L. (Giant Snapdragon) Begonia multiflora (Tuberous-rooted begonia) Begonia rex, Putz. (Begonia) cv. Thousand Wonders White Bougainvillea spectabilis Willd. (Bougainvillea) Callistephus chinensis (L.) Nees (China aster) Chrysanthemum sp. (Chrysanthemum) cv. Oregon Hibiscus Rosa-sinensis L. (Chinese hibiscus) Impatiens sultani, Hook. (Sultana) cv. White Imp	Dahlia variabilis Willd. (Dahlia) Fuchsia hybrida Voss (Fuchsia) Gardenia jasminoides Ellis (Cape Jasmine) Gardenia radicans Thunb. (Gardenia) Ixora coccinea L. (Ixora) Ligustrum lucidum Ait. (Ligustrum) Petunia X hybrida Hort. Vilm.-Andr. (Common Garden Petunia) Pittosporum tobira Ait. (Japanese pittosporum) Rhododendron catawbiense Michx. (Catawba rhododendron)	Carissa carandas L. (Carissa) Codiaeum variegatum Blume (Croton) Chrysanthemum leucanthemum L. (Daisy) Convallaria majalis L. (Lily-of-the-valley) Erica carnea L. (Spring heath) Gladiolus communis L. (Gladiolus) Erica sp. (Heath) Hosta sp. (Plantain lily) Juniperus conferta Parl. (Shore juniper) Rhododendron sp. (Alaska)

TABLE 12-4 (continued)

Plant Type	Susceptible	Intermediate	Tolerant
	Lathyrus odoratus L. (Sweet pea) Lupinus augustifolius L. (Lupine) Nerium oleander L. (Oleander) Pyracantha coccinea Roem. (Fire thorn) Rhododendron canescens [Michx.] Sweet (Hoary Azalea) Rosa sp. (Rose) Vinca minor L. (Periwinkle) cv. Bright Eyes		
Trees & Shrubs	Betula pendula Roth. (European white birch) Melaleuca leucadendra (L.) L. (Brittlewood)	Acer platanoides L. (Norway maple) Acer palmatum Thunb. (Japanese maple) Tilia grandiflora (Summer) Tilia cordata Mill. (Small-leaved European linden)	Carpinus betulus L. (European hornbean) Fagus sylvatica L. (Beech) Fagus sylvatica atropurpurea Kirchn. (Purple-leaved beech) Ginyko biloba L. (Gingko) Quercus robur L. (English oak) Robinia pseudoacacia L. (Black locust) Sambucus nigra L. (European elder) Ulmus glabra Huds. (Scotch elm) Ulmus montana With. (Mountain elm)
Weeds	Brassica sp. (Mustard) Helianthus annuus L. (Common Sunflower)	Malva parviflora L. (Cheeseweed) Stellaria media [L.] Cyrill (Chickweed) Taraxacum officinale Weber (Dandelion)	Amaranthus retroflexus L. (Pigweed) Chenopodium album L. (Lamb's-quarters) Chenopodium sp. (Neetle-leaved goosefoot)

they are based on subjective criteria obtained from several sources. Most of the classifications are developed from experimental fumigations conducted at various locations, at different times of the year, under different environmental conditions using different NO_2 exposure concentrations. Methods for assessing injury, such as percentage of leaves injured, amount of leaf surface affected, defoliation, etc. also varied. Therefore, a plant species considered tolerant by one investigator may be considered susceptible by another. Also, the interaction between genetic and environmental factors that control plant sensitivity is such, that given different sets of environmental parameters or other variables, the relative NO_2 sensitivity of species or cultivars within species can change.

A given plant and its individual leaves, will vary in sensitivity to NO_2, depending on the stage of development. In tobacco (Nicotiana sp.) the oldest leaves became chlorotic, middle age leaves became chlorotic with necrotic lesions, and injury to the younger leaves was limited to necrosis (VanHaut and Stratmann, 1967). In Ixora (Ixora coccinea) (MacLean et al., 1968) mustard (Brassica sp.), (Benedict and Breen, 1955) and tobacco (Nicotiana glutinosa) (Benedict and Breen, 1955) the older leaves were more sensitive. In other species such as chickweed (Stellaria media), dandelion (Taraxacum officinale), and pigweed (Amaranthus retroflexus) the middle age leaves were more sensitive. However, in sunflower (Helianthus annuus) middle age and older leaves respond similarly (Benedict and Breen, 1955). In citrus (Citrus sp.) necrosis was most severe on the youngest leaves (MacLean et al., 1968). Emerging or elongating needles of conifers were more susceptible than mature needles (Van Haut and Stratmann, 1967).

Only a few studies have reported the influence of edaphic factors on plant sensitivity to NO_2. Increasing soil moisture increased sensitivity in several weed and vegetable species (Benedict and Breen, 1955; Kato et al., 1974a).

Because of a possible relationship between atmospheric NO_2 and nitrogen metabolism, the influence of soil nitrogen on the plant response to NO_2 has been studied. Rogers et al. (1979) was unable to show differences in NO_2 uptake in corn (Zea mays) or soybean (Glycine max) plants that were grown in soil with varying levels of nitrogen. In contrast, Srivastava et al. (1975) reported that NO_2 uptake in bean (Phaseolus vulgaris) decreased with increasing levels of soil nitrogen. They also reported that NO_2-induced foliar injury decreased with increasing levels of soil nitrogen. Similar results were found in studies of other vegetables (Kato et al., 1974a; Kato et al., 1974b). Zahn (1975) reported that increasing the available soil nitrogen reduced NO_2-induced foliar injury. However, Troiano and Leone (1974) found that tobacco (Nicotiana glutinosa) grown on a low level of soil nitrogen was more resistant than when grown on a higher level of soil nitrogen. Kato et al. (1974a, 1974b) reported that plants grown on NH_4-N source were more sensitive to NO_2-induced injury and contained higher levels of foliar nitrite after NO_2 exposure than plants grown on a NO_3-N source. Kidney beans (Phaseolus vulgaris) and sunflower (Helianthus annuus) were grown on either ammonium, nitrate, nitrite or minus nitrogen sources. The plants that received either nitrate or nitrite through the nutrient solution contained less nitrite in the foliage following a 3-hour exposure to 7.52 mg/m^3 (4 ppm) NO_2 than plants grown on minus nitrogen or ammonium (Yoneyama et al., 1979). Similarly, tomato (Lycopersicon esculentum) and sunflower (Helianthus annuus) were grown at three levels of nitrogen nutrition ranging from 26 to 260 ppm N (Matsumaru et al., 1979). The plants were exposed to 0.56 mg/m^3 (0.3 ppm) NO_2 for 2 weeks. Plant growth was depressed between 0 and approximately 21% with no clear trend between the nitrogen concentration in the growth media and growth reductions.

Nitrogen nutrition can influence the effects of NO on tomatoes (Anderson and Mansfield, 1979). The plants were grown on either low, medium, or high levels of soil nitrogen and exposed to a concentration range 0 to 0.98 mg/m^3 (0.8 ppm) NO for 50 days. At a low level of soil nitrogen, plant growth increased with increasing levels of NO. At the medium level of soil nitrogen, the 0.49 and 0.98 mg/m^3 (0.4 and 0.8 ppm) levels of NO significantly reduced growth. At the high level of soil nitrogen, all levels of NO reduced plant growth compared to the controls.

Several researchers have studied the effect of light and time of day on plant sensitivity to NO_2. Zahn (1975) noted that alfalfa (Medicago sativa) exposed to NO_2 during the night were injured more extensively than plants exposed during the day. These findings were supported by Czech and Nothdurft (1952) who discovered that the toxic dose for 1-hour exposures of sugar beets (Beta vulgaris) was 10 times greater in the day time, 188 mg/m^3 (100 ppm) than at night, 18.8 mg/m^3 (10 ppm). Van Haut and Stratmann (1967) found leaf injury in oats (Avena sativa) was greater at night, but they also reported that once injury was initiated, the development of necrotic lesions was more rapid on warm sunny days. Kato et al. (1974a,b) determined that vegetable plants were severely injured from NO_2 exposures in the dark and contained higher foliar levels of nitrite than plants exposed in the light. Taylor (1968) reported that bean (Phaseolus vulgaris) plants were injured by 5.6 mg/m^3 (3 ppm) NO_2 in the darkness and that this dose caused as much damage as 11.3 mg/m^3 (6 ppm) NO_2 in the light. Zeevaart (1976) exposed 9 plant species to NO_2 in both light and dark. Eight species exhibited more damage when exposed in the dark. Injury was associated with an increase in nitrite and a decrease in expressed cell sap pH; fumigation of the plants with $NH_3 + NO_2$ reduced injury. However, with tobacco (Nicotiana glutinosa) light was required for injury to develop and there was no association between injury and either nitrite or cell sap pH. In studies with several vegetable plants, Inden (1975) showed that plants were most sensitive when exposed to NO_2 in the dark. Also leaves treated with the photosynthetic inhibitor DCMU were very sensitive to NO_2 exposures in the light. Plant susceptibility also varies at different times during the day. In a series of 2-hr exposures beginning at 0800-1000 hours and ending at 2000-2200 hours, injury to rye (Secale cereale) plants was greatest at mid-day (Van Haut and Stratmann, 1967).

12.3.2 Mode of Action

Since NO_2-induced perturbations occur at cellular sites within mesophyll tissue, NO_2 uptake into the leaf is required. Absorption is governed by factors regulating gaseous exchange between the atmosphere and the leaf (Nobel, 1974). The NO_2 diffuses from the boundary layer--bulk air interface and terminates with extraction onto mesophyll cell surfaces. The driving force for NO_2 uptake is a concentration gradient and net movement along this gradient is impeded by several leaf resistances: boundary layer, stomatal and mesophyll (residual) resistance.

Rodgers et al. (1979) suggested that NO_2 uptake in corn (Zea mays) and soybean (Glycine max) was directly related to stomatal resistance in the NO_2 concentration range of 0 to 1.09 mg/m^3 (0.58 ppm). Also, NO_2 uptake increased with light intensity through the action of light on stomatal resistance. In studies with beans (Phaseolus vulgaris) and higher NO_2 concentrations over the range of 0 to 13.16 mg/m^3 (7 ppm), NO_2 uptake was controlled more by internal leaf factors (mesophyll resistance) than stomatal resistance (Srivastava et al., 1975a, 1975b); NO_2 uptake was also reported to increase with concentration and decline with increasing exposure time. The NO_2 uptake rate in the dark was approximately one-half the rate in the light. Sunflower (Helianthus annuus) leaves absorbed approximately 14% as much NO_2 in the dark as in the light (Yoneyama et al., 1979). Eventhough NO_2 uptake in the dark is lower, NO_2 exposures in the dark cause greater injury (see 12.2.1).

Nitrogen dioxide reacts in water to produce nitrate, NO_3^-, and nitrite NO_2^- in dilute solutions. Similar reactions would be expected when NO_2 dissolves in the aqueous layer surrounding leaf mesophyll cells. Plants contain the enzymes, nitrate reductase that reduces nitrate to nitrite, and nitrite reductase that reduces nitrite to ammonia which can then be metabolized into organic nitrogen compounds. The nitrate reductase is induced in plants by the presence of its substrate, nitrate. Zeevaart (1974) grew plants with ammonia as the sole nitrogen source and showed that NO_2 exposures induced nitrate reductase activity and enzyme activity increased with NO_2 concentration and duration of exposure. Yoneyama et al. (1979) showed that exposure to 7.52 mg/m^3 (4 ppm) NO_2 for 6 hours increased nitrite reductase activity from 2- to 3-fold.

Faller (1972) grew sunflowers (Helianthus annuus) on a nitrogen free media and exposed the plants to NO_2 for three weeks at NO_2 concentrations ranging from 0 to 6 mg/m^3 (3.2 ppm). The control plants showed severe signs of nitrogen deficiency and restricted growth. However, the symptoms of nitrogen deficiency decreased and plant growth increased as the NO_2 concentration increased up to 29 percent more than the control levels. Also the nitrogen content of the plants was significantly increased by the exposure. Results of this study show that plants can use atmospheric sources of nitrogen (i.e., NO_2) as their sole nitrogen source. Tomato (Lycopersicon esculentum), sunflower (Helianthus annuus), and corn (Zea mays) derived approximately 16, 22 and 14% of their nitrogen, respectively, from NO_2 when the plants were exposed to 0.56 mg/m^3 (0.3 ppm) for 2 weeks (Matsumaru et al., 1979). The absorption rate of NO_2, based on plant dry weight, showed little change with soil nutrition and ranged around approximately 0.8 mg/g dry weight/day for tomato and sunflower to 0.3 mg/g dry weight/ day for corn.

Zeevaart (1976) grew peas (Pisum sativum) with ammonia as the only nitrogen source. When exposed to NO_2, nitrate and nitrite accumulated in the leaves. At the beginning of the exposures the nitrate to nitrite ratio was near 1, but with time, nitrite accumulated in the leaves while nitrate did not, suggesting that nitrate was converted to another compound. This finding appears to be related to the induction of nitrate reductase (Zeevaart, 1976). Exposures also increased the soluble amino (NH_2) groups in several plants and the protein content increased by 10-20 percent. Troiano and Leone (1977) reported that NO_2 exposures increased the organic nitrogen content of tomato plants (Lycopersicon esculentum).

Matsushima (1972) found that exposure to 75.2 mg/m^3 (40 ppm) NO_2 for 16 hours stimulated amino acid synthesis (indicated by ^{14}C labeling) although there was little change in organic acid synthesis. Using very high concentrations of $^{15}NO_2$ (0.01-5 percent by volume) Durmishidze and Nutzsubidze (1976) showed that 10 species of decidious trees, 5 species of grasses and 5 species of coniferous trees readily assimilated isotopically-labeled $^{15}NO_2$ and incorporated it into amino acids. Spinach (Spinacia oleracea) was exposed to 7.52 mg/m^3 (4 ppm) $^{15}NO_2$ for 2.5 hours to determine the metabolic fate of the nitrogen from the NO_2 (Yoneyama and Sasakawa, 1979). Isotopically labeled nitrogen was incorporated into both amino acids and amides in a pattern suggesting that the nitrogen was assimilated through the glutamine sythethase/glutamate synthase pathway. Rogers et al. (1979) reported that the plants accumulated isotopically labeled nitrogen proportional to the ambient NO_2 concentration over at range of 0.19 to 0.75 mg/m^3 (0.1 to 0.4 ppm). Following a 3-hour exposure to 0.60 mg/m^3 (0.32 ppm) NO_2, over 97% of the absorbed $^{15}NO_2$ was incorporated into reduced nitrogen compounds. However, the majority of this nitrogen remained in foliage, with only 3-5% being exported to the roots.

When plants are exposed to high levels of NO_2 the injured leaves frequently exhibit a waxy or water-soaked appearance prior to necrosis. This suggests that cell membranes were disrupted, possibly beyond repair. Berge (1963) suggested that NO_2 may cause cellular plasmolysis implying damage to cellular membranes. He also noted a decline in starch grains.

Felmeister and colleagues (1970) discussed the propensity of NO_2 to attach to lipid monolayers, especially those monolayers containing unsaturated lipids. These lipids have been proposed to be components of biological membranes. The work of Estefan et al. (1970) suggested that the products of NO_2 action on lipid monolayers included both transient and stable free radicals. Because of the high concentrations used in their experiments, it is not apparent that a similar response would occur at ambient concentrations.

Wellburn et al. (1972) studied the effects of NO_2 exposures on the ultrastructure of chloroplasts in vivo. Broad bean (Vicia faba) plants were exposed for 1 hour to 1.9, 3.8, or 5.6 mg/m^3 (1.0, 2.0, or 3.0 ppm). The leaves were harvested immediately after exposure and prepared for electron microscopy. Examination of the chloroplasts showed that NO_2 caused a swelling of the thylakoids associated with the stroma. These swellings appeared to be reversible since thylakoid swelling was not observed in chloroplasts of leaves exposed to unpolluted air immediately following NO_2 fumigation.

Kändler and Ullrich (1964) demonstrated that there was a reduced amount of carotene and chlorophyll in leaves after acute NO_2 exposure. Some species of lichens exposed to 3.76 mg/m^3

(2.0 ppm) NO_2 for 6 hours had reduced chlorophyll content; a dose of 7.52 mg/m^3 (4.0 ppm) NO_2 for 6 hours reduced chlorophyll content even further (Nash, 1976). In contrast, Taylor and Eaton (1966) found that chlorophyll content increased following chronic exposures to NO_2. Similarly, Horsman and Wellburn (1975) found that 0.188 to 1.88 mg/m^3 (0.1 to 1.0 ppm) NO_2 applied to pea (Pisum sativum) seedlings increased their chlorophyll content 5 to 10 percent. They also noted the deeper green color and downward curving of the leaves observed by Taylor and Eaton (1966).

In vivo experiments performed by Hill and Bennett (1970) showed that both NO and NO_2 inhibited apparent photosynthesis of oat (Avena sativa) and alfalfa (Medicago sativa) plants at concentrations below those that cause foliar lesions. The threshold dose for this inhibition was 0.74 mg/m^3 (0.6 ppm) for NO and 1.13 mg/m^3 (0.6 ppm) for NO_2 in 90 minute fumigations, but the inhibition occurred faster for NO than NO_2. The NO_x-induced inhibition of photosynthesis was not permanent. The rate of recovery for a given NO-induced inhibition level was faster than for NO_2. Recovery from NO-inhibited photosynthesis was generally complete within 1 hour. Full recovery from NO_2-induced inhibition of more than 25 percent required more than 4 hours. However, complete recovery of non-foliar injured plants was noted consistently within 1 day following fumigation. In fumigations introducing both NO and NO_2 (1:1) simultaneously, the degree of inhibition of apparent photosynthesis was the same as the sum of that induced by each pollutant when introduced separately.

Capron and Mansfield (1976) found a reduction in the photosynthetic rate of tomato (Lycopersicon esculentum) plants exposed to 0.47 mg/m^3 (0.25 ppm) NO_2, 0.31 mg/m^3 (0.25 ppm) NO or higher concentrations over a 20-hour period. The effect of the two gases in combination was an additive inhibition of photosynthesis. Srivastava et al. (1975a) studied the effects of NO_2 on the gas exchange of the primary leaves of bean (Phaseolus vulgaris). Apparent photosynthesis and dark respiration were both inhibited by NO_2 concentrations between 1.88 and 13.5 mg/m^3 (1 and 7 ppm). The degree of inhibition increased with increasing NO_2 concentration and exposure time. In exposures to NO_2, transpiration rate was effected less than photosynthesis or respiration. Hence, it was proposed that the principal effects of NO_2 on leaf gas exchange occurred in the leaf mesophyll cells and not on the stomata (Hill and Bennett, 1970; Srivastava et al., 1975a).

12.3.3 Visible Symptoms of NO_2 Injury

No one visual symptom or set of symptoms reliably indicates plant exposure to NO_2. The diagnosis of injury resulting from NO_2 is often difficult (Applied Science Associates, Inc., 1976; Taylor and MacLean, 1970). The injury pattern may vary within a species, cultivar, age of leaf, season of year, and/or pollutant dose.

Acute foliar marking produced by high concentrations of nitrogen dioxide exposures are characterized by water-soaked lesions which appear first on the upper leaf surface, followed by rapid tissue collapse. With time these lesions extend through the leaf and produce small, irregular necrotic patches. Necrotic areas are usually white to tan or brown and resemble SO_2-induced symptoms. Lesions occur between the veins of all sensitive plants, and may be located anywhere on a leaf surface, but they are most prominent at the apex along the margins. In monocots, acute NO_2 exposures most often result in yellow to ivory to white necrosis that begins at or just below the tips of the leaf blades. Necrotic margins and striped necrotic lesions between the veins also occur. In most grains and grasses, injury from acute exposures affects the entire width of the leaf blade.

In conifers, acute NO_2 injury usually begins at the needle tips and progresses towards the base. The boundary between healthy and injured tissues is sharply delineated by a brown or red-brown band. Young emerging needles show NO_2 injury at the tips, whereas older needles may develop necrosis in the central or basal portions of the needles. Injured needles may drop prematurely.

Chlorosis is one symptom of chronic NO_2 exposure but is nonspecific and can be a symptom of injury caused by other pollutants. For many chronic exposures, chlorosis precedes the appearance of chronic lesions. Cereal, grains, and corn leaves often develop longitudinal chlorotic bands before necrosis develops. In monocots, chlorosis may occur as transition zones between healthy tissue and the necrotic tips. In some broad leaf plants, chlorosis from chronic NO_2 exposures begins with many small yellow-green areas on the leaf surface which may merge as exposure continues. In some species, chlorosis may be concentrated near the leaf margins.

12.3.4 Dose Response

Exposures to most pollutants, including NO_x, are usually classified arbitrarily as acute or chronic. In experimental fumigations, acute exposures are of short duration at high pollutant concentrations. Chronic exposures are for longer periods (usually intermittent, occasionally continuous) at low concentrations. The ranges of concentrations and durations of exposure (doses) for acute and chronic exposures have not been defined. Most botanic investigators would designate NO_2 exposures of 3 to 5 mg/m^3 (1.6 to 2.66 ppm) or greater for up to 48 hours as acute and those for longer periods at lower concentrations as chronic. However, these definitions do not apply in the field near sources of NO_x emissions. There, an acute exposure is any single exposure causing plant injury. The term "chronic" is applied to a series of exposures that result in injury where no single exposure has an effect by itself.

12.3.4.1 Foliar Injury--Thomas (1952) observed that leaves of plants growing near nitric acid factories often had brown and black spots near the leaf margins. This was an early indication that oxides of nitrogen may be phototoxic. Haagen-Smit (1951) was one of the first to recognize the importance of NO_2 in causing photochemical smog effects on vegetation. He found that NO_2 added to experimental fumigation mixtures caused plant injury similar to that caused by ozone, but he provided no information on the direct effects on plants. Subsequently, Haagen-Smit et al. (1952) tested NO_2 at 0.75 mg/m^3 (0.4 ppm) on 5 species but observed no injury. Middleton (1958), Middleton et al. (1958) and Thomas (1961) all recognized that NO_2 appeared in photochemically polluted atmospheres as a by-product of combustion, but they postulated that levels were and would continue to be too low to cause vegetation injury.

Korth et al. (1964) found that beans (*Phaselous vulgaris*), tobacco (*Nicotiana tabacum*), and petunia (*Petunia multiflora*) were not injured by 1.88 mg/m^3 (1.0 ppm) NO_2 for 2 hours.

Middleton et al. (1958) suggested that 5.64-7.52 mg/m^3 (3-4 ppm) for 8 hours was a threshold concentration that could cause visible injury on pinto bean (*Phaseolus vulgaris*) leaves.

Czech and Nothdurft (1952) fumigated agricultural and horticultural crops with NO_2 in the laboratory and in small greenhouses. Rape (*Brassica napus*), wheat (*Triticum satuvim*), oats (*Avena sativa*), peas (*Pisum sp*.), potatoes (*Solanum tuberosum*), and beans (*Phaseolus vulgaris*) showed little or no injury from 56.4 mg/m^3 (30 ppm) NO_2 for 1 hour. Alfalfa (*Medicago sativa*), sugar beets (*Beta vulgaris*), winter rye (*Secale cereale*), and lettuce (*Lactuca sativa*) showed some effects. Fujiwara (1973) reported that 37.6-94 mg/m^3 (20-50 ppm) NO_2 for 30 to 60 minutes injured most plants studied.

Heck (1964) fumigated cotton (*Gossypium hirsutum*), pinto beans (*Phaseolus vulgaris*) and endive (*Cicorium endivia*) under controlled conditions with 1.88 mg/m^3 (1.0 ppm) NO_2 for 48 hours and observed slight but definite spotting of leaves. There was no injury produced at 1.88 mg/m^3 (1.0 ppm) NO_2 for 12 hours. In another study, the same species were fumigated with 0.94, 3.76 and 6.58 mg/m^3 (0.5, 2.0, and 3.5 ppm) NO_2 for 21 hours. At exposures of 6.58 mg/m^3 (3.5 ppm) NO_2 mild necrotic spots appeared on cotton (*Gossypium hirsutum*) and bean (*Phaseolus vulgaris*) leaves and the endive leaves (*Cicorium endivia*) were completely necrotic.

Taylor and Cardiff (unpublished data, cited in Taylor et al., 1975) exposed field crops to NO_2 in sunlight chambers. They found that several field crops exposed to 18.88 mg/m^3 (10 ppm) NO_2 for 90 minutes suffered little or no injury; but in tomato, a 90-minute exposure to 28.20 mg/m^3 (15 ppm) increased the extent of injury by 90 percent. They concluded that the injury threshold for several field crops would be 18.80 to 28.20 mg/m^3 (10 to 15 ppm) NO_2 for 90 minutes.

MacLean et al. (1968) exposed 14 ornamental and 6 citrus species to NO_2 concentrations ranging from 18.8 to 470 mg/m^3 (10 to 250 ppm) for 0.2 to 8 hours. Necrosis occurred in the citrus species when the leaves were exposed to 376 mg/m^3 (200 ppm) for 4 to 8 hours or 470 mg/m^3 (250 ppm) for 1 hour. Nonspecific marginal and intercostal necrosis developed within 1 hour after exposure. Young citrus leaves wilted and abscised at some lower doses.

Heck and Tingey (1979) conducted a series of short term fumigations which exposed field and vegetable crops to various NO_2 concentrations. In one experiment 10 field and vegetable species were exposed to 15.04, 30.08, or 60.16 mg/m^3 (8, 16, or 32 ppm) NO_2 for 1 hour (Table 12-5). At 60.16 mg/m^3 (32 ppm) levels of NO_2, all species showed visual injury. However, at 15.04 mg/m^3 (8 ppm) NO_2 exposure, only brome grass (*Bromus inermis*) and tomato (*Lycopersicon esculentum*) exhibited foliar injury. In a second experiment, 22 crop species were given 9 different time and concentration treatments. Exposure duration varied from 0.5 to 7 hours and NO_2 concentrations ranged from 3.76 to 37.6 mg/m^3 (2-20 ppm) (Table 12-6). An important conclusion from these experiments was that the extent of injury was greatest when the NO_2 levels were high, even for short time periods. For example, cotton exposed 28.2 mg/m^3 (15 ppm) for 1-hour had an injury rate of 27 percent for the three most sensitive leaves. When cotton was exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 2-hours the comparable injury rate was 2 percent. Therefore dose is not always a good predictor of injury.

TABLE 12-5. ACUTE INJURY TO SELECTED CROPS AFTER A 1-HOUR EXPOSURE TO NITROGEN DIOXIDE (HECK AND TINGEY, 1979)

Plants (Common, Cultivar, Scientific)[d]	Injury Index		
	8 ppm	16 ppm	32 ppm
Tomato, Roma[b] (*Lycopersicon esculentum*)	1	48	100
Wheat, Wells[c] (*Triticum durum*)	0	47	90
Soybean, Scott[b] (*Glycine max*)	0	26	100
Tobacco, Bel W_3[b] (*Nicotiana tabacum*)	0	23	97
Bromegrass, Sac Smooth[b] (*Bromus inermis*)	2	17	97
Swiss Chard, Fordhook Giant[c] (*Beta vulgaris cicla*)	0	11	62
Tobacco, White Gold[b] (*Nicotiana tabacum*)	0	1	70
Cotton, Acala 4-42[c] (*Gossypium hirsutum*)	0	0	54
Beet, Perfected Detroit[c] (*Beta vulgaris*)	0	0	36
Orchard Grass, Potomac[c] (*Dactylis glomerata*)	0	1	18
Tobacco, Bel W_3[c]	0	0	5

[a] Plants were exposed in Cincinnati, Ohio.

[b] Plants were exposed in August with light intensity at 2200 ft-c, temperature 28°C, humidity 75 percent.

[c] Plants were exposed in January with light intensity at 1400 ft-c, temperature 21°C, humidity 70 percent.

[d] Scientific name is given only when plant is first listed.

TABLE 12-6. PERCENT LEAF AREA INJURED BY DESIGNATED DOSAGE OF NITROGEN DIOXIDE (HECK AND TINGEY, 1979)

Plants (Common, Cultivar, Scientific)	Dosage (ppm x hr) (ppm) (hr)	2.5 5 0.5	4 4 1	6 3 2	10 20 0.5	14 7 2	15 15 1	20 5 4	20 10 2	35 5 7
Oats, Clintland 64 (*Avena sativa*)		0	0	0	80	2	84	0	39	21
Radish, Cherry Belle (*Raphanus sativus*)		0	0	0	95	0	90	1	31	2
Bromegrass, Sac Smooth		0	0	0	69	0	50	1	26	0
Begonia Thousand Wonders* White[d], (*Begonia Rex*)		0	1	0	26	0	35	4	49	5
Chrysanthemum, Oregon[d] * (*Chrysanthemum* sp.)		1	1	1	34	0	41	4	25	1
Sultana, White Imp[d] * (*Impatiens sultani*)		0	0	0	51	0	26	0	24	0
Oats, 329-80[b] (*Avena sativa*)		2	2	1	32	1	18	9	14	14
Cotton, Paymaster (*Gossypium hirsutum*)		0	0	6	50	0	27	2	2	1
Wheat, Wells		3	2	1	31	3	34	3	2	1
Cotton, Acala 4-42		0	0	0	28	0	28	0	1	1
Periwinkle, Bright Eyes[d] * (*Vinca minor*)		0	0	0	13	0	20	1	23	1
Oats, Pendek[c] (*Avena sativa*)		1	2	0	39	0	2	1	2	2

TABLE 12-6 (continued)

Plants (Common, Cultivar, Scientific)	Dosage (ppm x hr)	2.5	4	6	10	14	15	20	20	35
	(ppm)	5	4	3	20	7	15	5	10	5
	(hr)	0.5	1	2	0.5	2	1	4	2	7
Broccoli, Calabreese (*Brassica oleracea botrytis*)		0	0	0	19	0	21	0	0	0
Tobacco, Bel B (*Nicotiana tabacum*)		0	0	3	18	0	17	0	0	0
Tobacco, White Gold		0	0	1	18	0	6	0	0	0
Tobacco, Bel W_3		0	0	6	15	0	2	0	0	0
Tobacco, Burley 21 (*Nicotiana tabacum*)		0	0	0	8	0	0	0	0	0
Corn, Pioneer 509-W (*Zea mays*)		1	0	0	1	0	1	0	0	0
Corn, Golden Cross (*Zea mays*)		0	0	0	0	0	0	0	0	2
Azalea, Alaska * (*Rhododendron*, sp.)[d]		0	0	0	0	0	1	0	0	0
Sorghum, Martin (*Sorghum*, sp.)		0	0	0	0	0	0	0	0	0
Cucumber, Long Marketer (*Cucumis sativus*)		0	0	0	0	0	0	0	0	0

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[a] Plants were exposed in Cincinnati, Ohio. Each value is the average of 4 replicate plants except as noted. Plants are listed in general order of sensitivity.

Injury estimates based on the average of the three most sensitive leaves except for plants indicated (*) when the estimate was based on the total leaves per plant.

Heck and Tingey (1979) summarized the foliar injury data from their acute NO_2 exposures using the following model:

$$C = A_o + A_1 I + A_2/T$$

C = Concentration ppm

A_o, A_1, A_2 = constants (partial regression coefficients) specific for pollutant, plant species and environmental conditions.

I = percent injury

T = Time hours

The model recognizes the separate influence of time and concentration and permits the development of three-dimensional injury response surfaces. The model was used to estimate the NO_2 exposure durations and concentrations necessary to produce injury on susceptible, intermediate, and tolerant plants at the threshold injury level (Table 12-7).

12.3.4.2 Growth--Czech and Nothdruft (1952) using 1-hour exposures to 1,880 mg/m^3 (1000 ppm) NO_2 reported that the fresh weight of sugar beet (Beta vulgaris) roots were one-third less than that of the control plants. Zahn (1975) summarized the results of chronic exposures of 10 plant species to 2-4 mg/m^3 (1.06-2.12 ppm) NO_2 for 213 to 1900 hours (Table 12-8). The effects ranged from a 37 percent yield reduction in endive to no effect in roses. Exposures to 1.13 mg/m^3 (0.6 ppm) NO_2 for 30 days reduced the growth of buckwheat (Fagopyrum esculentum) and eggplant (Solanum melongana) (Fujiwara, 1973). The same concentration for 51 days increased the yield of rice (Oryza sativa). Stratmann (personal communication, cited in Taylor et al., (1975) showed that bush bean (Phaseolus vulgaris) growth was slowed by exposure to 1.88 mg/m^3 (1.0 ppm) NO_2 for 14 days. He suggested that a likely threshold dose for injury would be 0.752 mg/m^3 (0.4 ppm) NO_2 over a prolonged time period.

Taylor and Eaton (1966) found reduced fresh and dry weights of unifoliar leaves of pinto bean (Phaseolus vulgaris) plants exposed to NO_2 at 0.62 mg/m^3 (0.33 ppm) for 10 and 19 days. Also, leaves from tomato plants (Lycopersicon esculentum) exposed for 10 and 22 days to 0.21 to 1.17 mg/m^3 (0.11 to 0.62 ppm) were usually significantly smaller than corresponding leaves from non-fumigated plants. One of the most comprehensive reports on the effects of NO_2 on growth and yield is that of Spierings (1971). Continuous exposures of tomato plants (Lycopersicon esculentum) to 0.47 mg/m^3 (0.25 ppm) during the entire growth period (128 days) reduced growth of leaves, petioles and stems. The crop matured slightly earlier and there were substantial decreases in fresh weight yield (22 percent), average fruit weight (12 percent), and the number of fruit (11 percent). After exposures to 0.94 mg/m^3 (0.5 ppm) for 10 days or 0.47 mg/m^3 (0.25 ppm) for 29 days, fumigated tomato plants were taller than the controls, but stems were smaller in diameter, leaves were not as large, and the fresh weights of the plants were less.

TABLE 12-7. PROJECTED NO_2 EXPOSURES THAT MAY INDUCE 5 PERCENT FOLIAR INJURY LEVELS ON SELECTED VEGETATION (HECK AND TINGEY, 1979)

Time (hr)	Concentrations Producing Injury					
	Susceptible[a]		Intermediate[a]		Tolerant[a]	
	ppm	m3/m3	ppm	m3/m3	ppm	m3/m3
0.5	6-10	11.28-18.80	9-17	16.92-31.96	≥ 16	≥ 30.08
1.0	4-8	7.52-15.04	7-14	13.16-26.32	≥ 13	≥ 24.44
2.0	3-7	5.64-13.16	6-12	11.18-22.56	≥ 11	≥ 20.68
4.0	2-6	3.76-11.28	5-10	9.40-18.80	≥ 9	≥ 16.92
8.0	2-5	3.76- 9.40	4-9	7.52-16.92	≥ 8	≥ 15.04

[a] Plant type.

TABLE 12-8. EFFECT OF CHRONIC NO_2 EXPOSURES ON PLANT GROWTH AND YIELD (ZAHN, 1975)

Plant Type	NO_2 Concentration (mg/m3)	Duration of Exposure (hours)	Effect
Wheat	2	334	No effect on grain yield, but the straw yield was reduced 12%.
Bush Bean	2	639	Yield reduced 27%; Some chlorosis
Endive	2	620	Yield reduced 37%
Carrot	4	357	Yield reduced 30%; Some chlorosis
Radish	4	278	Yield reduced 13%
Currant	2	213	Yield reduced 12%
Roses	4	357	No injury
European Larch	2	537	No injury
Spruce	2-3	1900	7% decrease in linear growth. Growth was decreased 17% in the year following the exposure.

[a] Necrosis did not occur on any plants.

Thompson et al. (1970) exposed navel orange trees to NO_2 continuously for 290 days. When compared to trees exposed to filtered air, those fumigated with NO_2 concentrations ranging from 0.12 to 0.47 mg/m^3 (0.06 and 0.25 ppm) showed a significant increase in fruit drop throughout the exposure period and a significant reduction both in number and weight of fruit at harvest. At Upland, California, ambient and twice ambient levels of NO_2 were added to carbon filtered air supplied to navel oranges (Citrus sinensis) with no effects on leaf drop or yield (Thompson et al., 1971). Recent studies suggested that 0.47 mg/m^3 (0.25 ppm) or less of NO_2 supplied continuously for 8 months will increase leaf drop and reduce the yield of navel oranges (Citrus sinensis) (Taylor et al., 1975).

To determine the effects of NO on the growth of tomatoes (Lycopersicon esculentum), four different tomato cultivars were exposed to 0.49 mg/m^3 (0.4 ppm) NO for 35 days (Anderson and Mansfield, 1979). In two cultivars, total weight, shoot weight and leaf areas was reduced, however, in the other cultivars, NO stimulated plant growth. Exposure of another tomato cultivar to 0.75 mg/m^3 (0.4 ppm) NO for 19 days reduced leaf area, leaf weight and stem weight (Capron and Mansfield, 1977). In the same study, 0.19 mg/m^3 (0.1 ppm) NO_2 had no effect on plant growth and the mixture of 0.19 mg/m^3 (0.1 ppm) NO_2 and 0.49 mg/m^3 (0.4 ppm) NO reduced growth to the same extent as the NO alone (Capron and Mansfield, 1977). Tomatoes grown at three levels of soil nitrogen and were exposed to a concentration range of 0 to 0.98 mg/m^3 (0.8 ppm) NO. At harvest (50 days), total weight, shoot weight, and leaf area were measured (Anderson and Mansfield, 1979). At a low level of soil nitrogen, NO stimulated plant growth as reflected in all growth parameters. At a medium level of soil nitrogen, plant growth was decreased at a concentration of 0.49 mg/m^3 (0.4 ppm) NO. At the high level of soil nitrogen, all levels of NO depressed plant growth.

Because of the inter-relationship between concentration and time, there is no single threshold dose for an effect. MacLean (1975) summarized the literature to illustrate the relationship between NO_2 concentration and duration of exposure (dose) for various effects (Figure 12-4). Three threshold curves are shown in Figure 12-5. These are approximate estimates, as reported in the literature, based on various responses of many plant species, to acute and chronic NO_2 doses. The threshold curve for NO_2 doses that result in the death of plants is short because it is based on limited information. NO_2 doses approaching this threshold result in complete defoliation of some species but are not lethal. The threshold curve for leaf injury is based on observations at many NO_2 doses. The shift in leaf injury from necrosis to chlorosis for NO_2 doses along this curve generally occurred between 10 and 100 hours. Because no measurable effects have been reported for NO_2 doses below the lower curve, it can be considered as the threshold for metabolic and growth effects. NO_2 doses in the area between this curve and the threshold curve for leaf injury are those that do not injure leaves but often result in growth suppression or effects on photosynthesis or other plant processes.

These thresholds (Figure 12-5), assuming that they are reasonable estimates for vegetation in general, can serve as points of reference to evaluate air quality standards for NO_2 in the

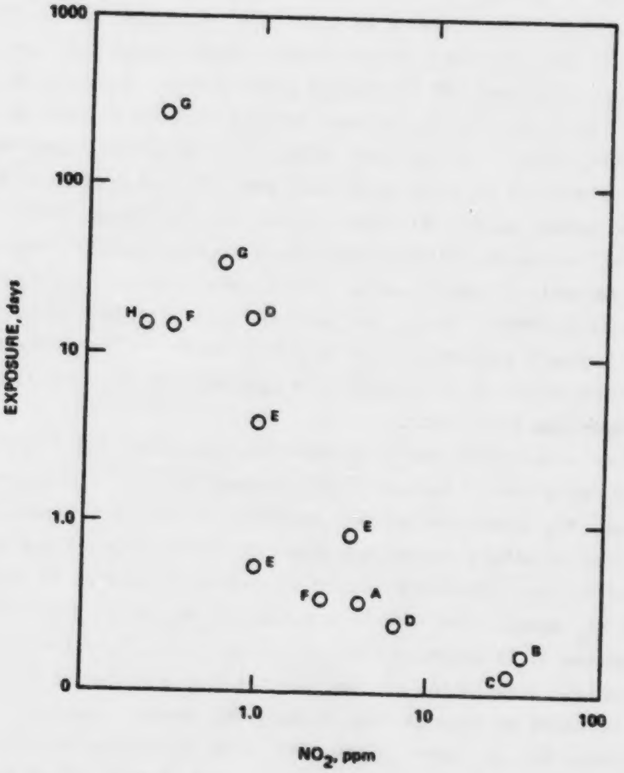

Figure 12-4. Summary of effects of NO_2 on vegetation. The points describe a dosage line above which injury was detected (Jordan, 1969). Individual points were taken from the following references: (A) Middleton et al., 1958; (B) Hill et al., 1974; (C) Czech and Northdurft, 1952; (D) H. Strattman (in Taylor et al., 1975); (E) Heck, 1964; (F) Taylor and Eaton, 1966; (G) Thompson et al., 1970; and (H) Matsushima, 1976.

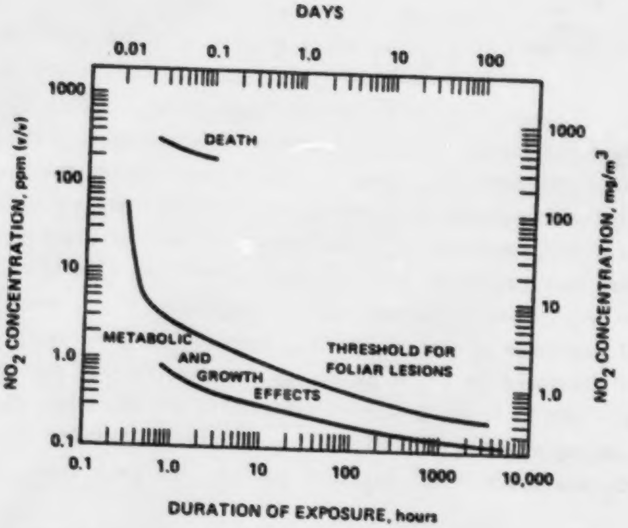

Figure 12-5. Threshold curves for the death of plants, foliar lesions, and metabolic or growth effects as related to the nitrogen dioxide concentration and the duration of exposure (McLean, 1975).

atmosphere in the absence of other gases and they can be viewed with respect to NO_2 concentrations that occur in the atmosphere.

12.3.5. Effects of Gas Mixtures on Plants

Mixtures of dissimilar pollutants often occur in nature. A typical combination includes NO_2 with sulfur dioxide (SO_2) and/or ozone (O_3). Reinert et al. (1975) reviewed information on these types of pollutant combinations. Earlier the assumption was made that NO_x at normal atmospheric concentrations was important only on the basis of its participation in the photochemical oxidant reactions. However, based on studies in which plants were exposed to combinations of pollutants including NO_2 it now appears that ambient concentrations of NO_2 in conjunction with other pollutants may have a direct effect on plants. The results of these studies are described.

12.3.5.1 *Nitrogen Dioxide and Sulfur Dioxide*--To determine the impact of ambient air pollution on vegetation, NO_2 and SO_2 have been evaluated for their combined effects.

Tingey et al. (1971) found that neither 3.76 mg/m^3 (2.0 ppm) NO_2 nor 1.31 mg/m^3 (0.5 ppm) SO_2 alone caused foliar injury. However, a mixture of 0.188 mg/m^3 (0.10 ppm) NO_2 and 0.262 mg/m^3 (0.10 ppm) SO_2 administered for 4 hours caused foliar injury to pinto bean (*Phaseolus vulgaris*), radish (*Raphanus sativus*), soybean (*Glycine max*), tomato (*Lycopersicon esculentum*), oat (*Avena sativa*) and tobacco (*Nicotiana tabacum*). Exposure to 0.282 mg/m^3 (0.15 ppm) NO_2 in combination with 0.262 mg/m^3 (0.1 ppm) SO_2 for 4-hours caused greater foliar injury. Traces of foliar injury were observed at 0.094 mg/m^3 (0.05 ppm) NO_2 and 0.131 mg/m^3 (0.05 ppm) SO_2.

Matsushima (1971) observed more leaf injury on several plant species from a mixture of NO_2 and SO_2 than that caused by each pollutant alone. He also tested different sequences of exposure. When NO_2 exposure preceded SO_2, the degree of injury was similar to that resulting from individual exposures to either gas. But when SO_2 exposure was followed by NO_2 the degree of leaf injury increased as would be typical of simultaneous exposures to both pollutants. Fujiwara et al. (1973) found greater-than-additive effects when peas (*Pisum sativum*) were exposed to 0.188 mg/m^3 (0.1 ppm) NO_2 in combination with 0.262 mg/m^3 (0.1 ppm) SO_2. When 0.376 mg/m^3 NO_2 and 0.524 mg/m^3 SO_2 (0.2 ppm of each gas) were used, the effect was only additive.

When a large number of desert species were exposed to either SO_2 or combinations of SO_2 and NO_2 (ratio approximately 4:1) injury from SO_2 and mixtures of $SO_2 + NO_2$ was similar (Hill et al., 1974). NO_2 decreased the foliar injury threshold of SO_2 on tomatoes (*Lycopersicon esculentum*), geranium (*Pelargonium sp.*), and petunia (*Petunia sp.*) (de Cormis and Luttringer, 1976). Exposure to 0.79 mg/m^3 (0.3 ppm) SO_2 caused no foliar injury but the same concentration of SO_2 in conjunction with 0.94 mg/m^3 (0.5 ppm) NO_2 caused foliar injury. Injury from the gas mixture increased with increasing humidity.

Observations in the vicinity of an arsenal emitting low concentrations of both NO_2 and SO_2 found foliar injury on several conifer species which was attributed to the interaction of the 2 gases (Skelly et al., 1972). The maximum observed 1-hour concentration of NO_x was 0.585

ppm and 2-hour maximum for SO_2 was 0.670 ppm. Effects on growth rate of two tree species was correlated with production activities at the arsenal over a 30 year period suggesting that mixtures of NO_2 and SO_2 may reduce plant growth (Stone and Skelly, 1974).

Bennett et al. (1975) studied the effects of NO_2 and SO_2 mixtures on radish (*Raphanus sativus*), swiss chard (*Beta vulgaris*), oats (*Avena sativa*) and peas (*Pisum sativum*). Treatments consisted of 1- and 3-hour fumigations with the pollutants separately and with SO_2 and NO_2 (1:1) mixtures in concentrations ranging from 0.33-2.62 mg/m^3 SO_2 and 0.23-1.88 mg/m^3 NO_2 (0.125 to 1.0 ppm). No visible injury occurred on experimental plants treated with NO_2 alone or from exposures to SO_2 concentrations of less than or equal to 1.31 mg/m^3 (0.5 ppm). The minimum exposure doses which caused visible injury to radish leaves were 1-hour exposures to a mixture of 0.94 mg/m^3 NO_2 and 1.31 mg/m^3 SO_2 (0.5 ppm of each gas) or to 1.95 mg/m^3 (0.75 ppm) SO_2 alone. The data indicated that SO_2 and NO_2 in combination may enhance the phytotoxicity of these pollutants, but relatively high doses were required to cause injury.

Reinert et al. (1975) summarized the effects of gas mixtures on foliar injuries to a number of crops (Table 12-9). The data indicate that ambient concentrations of NO_2 and SO_2 may interact to injure vegetation.

A study was conducted to determine the effects of low concentrations of NO_2 and SO_2 singly and in combination on the growth of four grass species (Ashenden, 1979a; Ashenden and Mansfield, 1978; Ashenden and Williams, 1980). Plants were grown and exposed in four small greenhouses which received either charcoal filtered air, 0.21 mg/m^3 (0.11 ppm) NO_2, 0.29 mg/m^3 (0.11 ppm) SO_2 or a mixture of both gases at these same concentrations. The plants were exposed for 103.5 hours per week, which resulted in weekly mean concentrations of 0.13 mg/m^3 (0.068 ppm) NO_2 and 0.18 mg/m^3 (0.068 ppm) SO_2. The plants were harvested monthly and various growth parameters were measured. The results of the experiments are summarized in Table 12-10. Nitrogen dioxide significantly reduced growth parameters of orchard grass and Kentucky bluegrass, but had no effect or slightly stimulatory effect on the growth of Italian ryegrass and timothy. However, growth parameters of all the species were reduced by SO_2. The combination of NO_2 and SO_2 significantly reduced the growth parameters of all species tested and many of the effects were determined to be synergistic. These data were collected during the winter when the plants were in a period of slow growth which may have increased the pollutant's toxicity. However, the data clearly show that intermittent exposures to ambient concentrations of NO_2 and SO_2 singly and in combination can significantly depress yield parameters of important forage grasses.

Alfalfa (*Medicago sativa*) exhibited a greater-than-additive response, i.e., a greater inhibition of apparent photosynthesis (CO_2 uptake) when NO_2 and SO_2 were applied together for 2 hours at 0.47 mg/m^3 (0.25 ppm) NO_2 and 0.655 mg/m^3 (0.25 ppm) SO_2 (White et al., 1974). A mixture of 0.282 mg/m^3 (0.15 ppm) NO_2 and 0.393 mg/m^3 (0.15 ppm) SO_2 for 2-hours decreased apparent photosynthesis 7 percent more than when the total of the two gases was applied independently. At higher concentrations, 0.5 ppm of each gas, the effects were not greater-than-

TABLE 12-9. PLANT RESPONSE TO SULFUR DIOXIDE AND NITROGEN DIOXIDE MIXTURES
(TINGEY ET AL., 1971; MATSUSHIMA, 1971; BENNETT ET AL., 1975; HILL ET AL., 1974)

Plant Species	Exposure Chamber[a,c]	SO_2/NO_2 (ppm)[2]	Exposure duration (hours)	Plant Response (% injury)	Mixture Response[b,c]	Plant Age (weeks)
Avena sativa L.	CE	0.75/0.75	1 or 3	0-5	+	4-5
Beta vulgaris var. cicla L.	CE	0.75/0.75	1 or 3	0-5	+	4-5
Lathyrus odoratus L.	CE	0.75/0.75	1 or 3	0-5	+	4-5
Raphanus sativus L.	CE	0.75/0.75	1 or 3	5-8	+	4-5
A. sativa	CE	0.15-0.25/0.1-0.2	4	0	*	2-3
R. sativus	CE	0.15-0.25/0.1-0.2	4	0	*	2-3
Phaseolus vulgaris L.	CE	0.15-0.25/0.1-0.2	4	0	*	2-3
R sativus	CE	0.5/0.5	1 or 3	0-5	+	4-5
Nicotiana tabacum L.	GH	0.1/0.1	4	0-10	+	7-8
Orzopsis hymendoides (R&S) Ricker	F	0.5-0.7/0.15-0.21	2	16	0	*
Populus tremuloides Michx.	F	0.5-0.7/0.15-0.21	2	1	0	*
Sphaeralcea munroana Spach.	F	0.5-0.7/0.15-0.21	2	31	0	*
P. vulgaris	*	1.5/15	1.17	70-75	+	3-4
Lycopersicon esculentum Mill.	*	2.3/13	1	35-85	+	3-5
Cucumis sativus L.	*	2.3/12	0.67	50-100	+	3-4
A. sativa	*	2.4/13	1	40-75	-	3-4
Capsicum frutescens L.	*	2.4/15	1	10-58	+	5-6
P. vulgaris Pinto	GH	0.05-0.25/0.05-0.25	4	0-24	+	3-4
A. sativa	GH	0.05-0.25/0.05-0.25	4	0-27	+	3-4
R. sativus	GH	0.05-0.25/0.05-0.25	4	0-27	+	3-4
Glycine max (L.) Merr.	GH	0.05-0.25/0.05-0.25	4	0-35	+	3-4
N. tabacum	GH	0.05-0.25/0.05-0.25	4	0-18	+	7-8
L. esculentum	GH	0.05-0.25/0.05-0.25	4	0-17	+	5-6

[a] CE, Control environment; GH, greenhouse; F, field.

[b] +, Greater than additive; 0, additive; -, less than additive.

[c] * Not defined.

TABLE 12-10. THE EFFECTS OF NO_2 AND SO_2 SINGULARLY AND IN COMBINATION ON THE GROWTH OF SEVERAL GRASSES[1]

Response	Species[2]	Pollutant[3]			Effect Synergistic
		NO_2	SO_2	$NO_2 + SO_2$	
Leaf Area	Orchard grass	21	5	72*	yes
	Kentucky bluegrass	17	28*	84*	yes
	Italian ryegrass	1+	22	43*	yes
	Timothy	30+	11	82*	yes
Number of Tillers	Orchard grass	1	10	32*	yes
	Kentucky bluegrass	9	27*	61*	yes
	Italian ryegrass	17	23*	32*	
	Timothy	6	33*	55*	
Dry Weight green leaves	Orchard grass	7	28*	83*	yes
	Kentucky bluegrass	29*	39*	88*	
	Italian ryegrass	10	28*	65*	yes
	Timothy	14+	25*	84*	yes
Dry Weight dead leaves and stubble	Orchard grass	46*	52*	67*	
	Kentucky bluegrass	27*	37*	57*	
	Italian ryegrass	5	3+	28*	
	Timothy	12	47*	64*	
Dry Weight roots	Orchard grass	11	37*	85*	yes
	Kentucky bluegrass	47*	54*	91*	
	Italian ryegrass	35+	7+	58*	yes
	Timothy	1+	58*	92*	

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[1] Data are expressed as percent reductions from the control and are derived from references 127a and 127b. The exposures were for 20 weeks (140 days). Plants were exposed for 103.5 hr/week. The concentrations of NO_2 and SO_2 during exposure were 0.11 ppm which resulted in a weekly average concentration of 0.068 ppm.

[2] The scientific names are: Orchard grass, *Dactylis glomerata*; Kentucky bludgrass, *Poa pratensis*; Italian ryegrass, *Lolium multiforum*; Timothy, *Phleum pratense*.

[3] Numbers followed by + indicate increase above the control and * indicates significant reductions of the 5% significance level or greater.

additive. Exposures of alfalfa (Medicago sativa) to 0.62 mg/m^3 (0.33 ppm) NO_2 and 2.62 mg/m^3 (1 ppm) SO_2 at an ambient CO_2 concentration for 1 to 3 hours reduced the photosynthetic rate approximately 50% (Hou et al., 1977). When the ambient carbon dioxide concentration was increased 645 ppm, the inhibitory effect of NO_2 and SO_2 on photosynthesis was only 50% as large as at ambient CO_2 levels. In studies with pea (Pisum sativum) Bull and Mansfield (1974) reported that over the concentration range of 0-0.47 mg/m^3 (0.25 ppm) NO_2 and 0-0.655 mg/m^3 (0.025) SO_2, photosynthesis was inhibited. The duration of exposure was not given. The effect of the two gases was additive in inhibiting of photosynthesis. In bean (Phaseolus vulgaris) when 0.188 mg/m^3 (0.10 ppm) NO_2 and 0.262 mg/m^3 (0.10 ppm) SO_2 were applied individually the pollutants stimulated short-term increases in transpiration, but the combination of NO_2 and SO_2 decreased the transpiration rate (Ashenden, 1979b).

Horsman and Wellburn (1975) studied the effects of NO_2 and SO_2 mixtures on several enzyme systems in peas (Pisum sativum). Peroxidase activity was enhanced somewhat by SO_2 alone but not by NO_2. However, 0.188 mg/m^3 (0.1 ppm) NO_2 plus 0.524 mg/m^3 (0.2 ppm) SO_2 for 6 days increased the activity by 24 percent. A 100 percent increase occurred when 0.188 mg/m^3 (0.1 ppm) NO_2 plus 5.24 mg/m^3 (2.0 ppm) SO_2 was used for 6 days. The effect was much greater-than-additive. The increase in peroxidase activity was considered a typical stress response of the plant. Similar studies have shown that glutamate dehydrogenase activity is stimulated as a greater-than-additive response by mixtures of NO_2 and SO_2 (Wellburn et al., 1976).

12.3.5.2 Nitrogen Dioxide with Other Pollutants--Matsushima (1971) reported that combinations of NO_2 and O_3 were less injurious to pepper (Capsicum furtesceans) and tomato (Lycopersicon esculentum) than similar concentrations of either gas alone. Loblolly pine and American sycamore (Platanus occidentalis) were exposed to either 0.10 mg/m^3 (0.05 ppm) ozone and/or 0.19 mg/m^3 (0.10 ppm) NO_2 for 6 hours per day for 25 days and effects on injury and growth were determined. At this concentration NO_2 had no deletrious effects on plant growth or injury. The combination of O_3 plus NO_2 yielded the same results as the effects of ozone alone. Reinert and Gray (1977) obtained different results using different pollutant combinations with radish (Raphanus sativus), pepper (Capsicum protescerus) and tomato (Lycopersicon esculentum). They found that NO_2, SO_2 or O_3 were less injurious when used individually than when in combinations of NO_2 + SO_2, NO_2 + O_3, or SO_2 + O_3. de Cormis and Luttringer (1976) found that a mixture of 0.31 mg/m^3 (0.12 ppm) SO_2, 0.56 mg/m^3 NO_2 (0.3 ppm) and 0.2 mg/m^3 (0.1 ppm) O_3 caused extensive leaf necrosis in tomato (Lycopersicon esculentum) within 2 hours.

It is clear from these limited data that levels of NO_2 generally considered below the injury threshold may interact with other common air pollutants to induce vegetation injury. Extensive research is needed to verify this phenomenon under field conditions. It appears that concentrations of NO_2 between 0.188 mg/m^3 (0.1 ppm) to 0.47 mg/m^3 (0.25 ppm) can cause direct effects on vegetation in combination with certain other pollutants.

12.4 SUMMARY

12.4.1 Effects on Ecosystems

Ecosystems represent the natural order by which living organisms are bound to each other and to their environment. They are, therefore, essential to the existence of any species on earth, including man, and as life support systems their value cannot be quantified in economic terms.

Ecosystems are important in the production of food, in the regeneration of essential nutrients as well as atmospheric components, in the assimilation or breakdown of many pollutants from the air, water, and soil, and in energy flow. They also give aesthetic pleasure and improve the quality of life.

The nitrogen cycle, an ecosystem function, is essential for all life because nitrogen is necessary in the formation of all living matter. Man has influenced the cycling of nitrogen by injecting fixed nitrogen into the environment or contributing other nitrogenous compounds which perturb the cycle.

Human activities have unquestionably increased the amounts of nitrates and related compounds in some compartments of the environment. The effects of such increased concentrations of nitrogen compounds may be beneficial or adverse, or both. Effects of both kinds may occur simultaneously, and may be felt in media or in ecological compartments quite removed from those that initially receive anthropogenic nitrogenous inputs.

Assessment of the influence of nitrogen oxides on ecosystems is complicated by several factors. Nitrogen oxides: (1) react with abiotic components of the natural environment as well as with individual organisms; (2) react with varying numbers of dissimilar populations within ecosystems; and (3) may suppress individual populations and thus affect ecosystem functioning.

One function of ecosystems is the cycling of nutrients such as nitrogen. Any effect, environmental or biological, which interferes with the recycling process could have a deleterious effect on the total ecosystem.

At the present time there are insufficient data to determine the impact of nitrogen oxides as well as other nitrogen compounds on terrestrial plant, animal or microbial communities. It is possible, however, to estimate the approximate magnitude of anthropogenic nitrogen fluxes to ecosystems, using the limited amount of monitoring data available or mass balance calculations. Such estimates, and quantitative information about the nitrogen cycle at specific sites in the system under study, make it possible to reach some conclusions about the possible ecological significance of the added nitrogen. In addition, where the data base is more extensive, as it is for a number of lakes in various stages of eutrophication, more quantitative dose-response relationships can be estimated.

A reduction in diversity within a plant community results in a reduction in the amount of nutrients present so that growth of remaining individuals decreases.

Pollutants act as predisposing agents so that disease, insect pests and abiotic forces can more readily injure the individual members of ecosystems. The loss of these individuals results in reduction in diversity and simplification of an ecosystem.

12.4.2 Effects on Vegetation

Sensitivity of plants to NO_2 varies with species, time of day, light, stage of maturity, type of injury assayed, soil moisture, and nitrogen nutrition.

When exposures to NO_2 alone are considered, the ambient concentrations that produce measurable injury are higher than those that normally occur in the United States (Chapter 8). Tomato (*Lycopersicon esculentum*) plants exposed continuously to 0.47 mg/m^3 (0.25 ppm) for 128 days were reduced in growth and suffered a decreased yield of 12 percent. Leaf drop and reduced yield occurred in naval oranges exposed to 0.47 mg/m^3 (0.25 ppm) continuously for 8 months. Pinto beans (*Phaseolus vulgaris*), endive (*Cicorium endivia*) and cotton (*Gossypium hirsutum*) exhibited slight leaf spotting after 48 hours of exposure to 1.88 mg/m^3 (1.0 ppm). Reduced growth in bush beans (*Phaseolus vulgaris*) was reported after a 14-day exposure to 1.88 mg/m^3 (1.0 ppm). Other reports cited no injury in beans (*Phaseolus vulgaris*), tobacco (*Nicotiana tabacum*), or petunia (*Petunia multiflora*) after a 2-hour exposure of the same concentration.

Exceptions to this generality, however, have been observed. For example, the growth of Kentucky bluegrass was significantly reduced (approximately 25%) by exposures to 0.21 mg/m^3 (0.11 ppm) NO_2 for 103.5 hours per week for 20 weeks during the winter months. Similar exposures to other grass species generally had no deletrious effect on plant growth.

Nitrogen dioxide concentrations ranging from 0.188 to 1.88 mg/m^3 (0.1 to 1.0 ppm) increased chlorophyll content in pea (*Pisum sativum*) seedlings from 5 to 10 percent. The significance of the increased chlorophyll is unknown. Some species of lichens, a plant sometimes used as an indicator of the presence of phytotoxic gases, exposed to 3.96 mg/m^3 (2.0 ppm) for 6 hours showed a reduced chlorophyll content.

In contrast to the studies cited on the effects of NO_2 alone, a number of studies on mixtures of NO_2 with SO_2 showed that the NO_2 injury threshold was significantly decreased and that the effects of the two gases in combination were at least additive and usually greater-than-additive. Concentrations at which observable injury occurred were well within the ambient concentrations of NO_2 and SO_2 occurring in some areas of the United States. Neither 3.96 mg/m^3 (2.0 ppm) NO_2 nor 1.31 mg/m^3 (0.5 ppm) SO_2 alone caused foliar injury. Research data from grass species exposed for 20 weeks to concentrations of 0.21 mg/m^3 (0.11 ppm) NO_2 and 0.29 mg/m^3 (0.11 ppm) SO_2 for 103.5 hours per week showed significant reductions in yield parameters ranging from 30 to 90% indicating that concentrations of these two gases occurring simultaneously can have major deletrious effects on plant growth.

CHAPTER 12

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13. EFFECTS OF NITROGEN OXIDES ON MATERIALS

The damaging effects of atmospheric nitrogen oxides (NO_x) have been established for a variety of materials, including dyes, fibers, plastics, rubber and metals. Other atmospheric components which can damage materials include particulates, sulfur dioxide and oxidants (ozone).

These effects occur through chemical changes which result in lowered material performance or service. This causes consumer disappointment and economic losses, both to the product manufacturer, and to the nation at large. The most injurious nitrogen oxide is nitrogen dioxide (NO_2). This chapter presents an evaluation of the effects of oxides of nitrogen on textile dyes, man-made and natural fibers, plastics, elastomers and metals.

It should be pointed out that some exposure situations described in this chapter, which lead to an economic loss (mostly involving textiles), characteristically take place indoors. Although indoor and outdoor pollutant concentrations are not always directly related or proportional, it is reasonable to expect that much of the pollution in indoor environments comes from ambient air. Indoor sources such as gas appliances in homes or combustion-powered fork lifts in warehouses may also contribute to indoor pollutant levels.

13.1 EFFECTS OF NITROGEN OXIDES ON TEXTILES

The types of damage to textiles attributed to NO_x action include:

- Fading of dyes on cellulose acetate (also known as acetate and cellulose acetate rayon), cotton, viscose rayon (Upham and Salvin, 1975), and nylon.
- Color changes on permanent press garments containing polyesters.
- Yellowing of white fabrics.

13.1.1 Fading of Dyes by Nitrogen Oxides

13.1.1.1 Fading of Dyes on Cellulose Acetate--The NO_x fading of acetate, dyed blue, or in shades in which blue is a component, results in pronounced reddening. Rowe and Chamberlain (1937) demonstrated that the causative factors were nitrogen oxides in combustion gases. The blue dyes which were and are still in widespread use are derivatives of anthraquinone.

Blue dyes, such as Disperse Blue 3, a dye commonly used to test for the presence of NO_x, contain amino groups which are susceptible to nitrosation and oxidation by NO_x. The fading of Disperse Blue 3 as a result of NO_x action is caused by the formation of a nitrosamine at the vulnerable alkylamine site(s), or the production of a phenolic group (-OH) at the amine site(s), through oxidation (Couper, 1951). Both of these reaction products have a red color, which is seen when certain fabric-dye combinations are exposed to NO_2.

Salvin et al. (1952) found that cellulose acetate is an excellent absorber of NO_2. Absorption characteristics of fibers also are believed to play an important role in dye-fading mechanisms. Polyester and polyacrylic fibers have low NO_2 absorption rates while nylon, cotton, viscose rayon and wool have intermediate rates. While cellulose acetate and cellulose triacetate have high NO_2 absorption rates, the NO_2 is released upon heating. Nylon and wool,

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materials which contain reactive amino groups, hold the NO_2 in chemical combination and release it upon hydrolysis. The oxides of nitrogen are retained by cotton and viscose rayon, fabrics collectively referred to as cellulosics.

Both blue and red dyes having the anthraquinone structure are susceptible to NO_x action. These dyes include Disperse Blue 7, Disperse Blue 3, Disperse Red 11 and Disperse Red 55. The fading of these dyes is recognized and noted in shade books published by dye suppliers.

Asquith and Campbell (1963) have noted that fading also occurs with certain yellow dyes of the diphenylamine class.

Dye fading associated with NO_2 exposure of cellulose acetate and cellulosics is summarized in Table 13-1. Testing methods predictive of dye fading have been summarized in the literature (American Association of Textile Chemists and Colorists, 1972; Hemphill, 1976; Salvin, 1974a; Seibert, 1940). Selected anthraquinone-b and blue dyes exhibit high resistance to fading by NO_x (Salvin, 1959; Seymour and Salvin, 1949; Straley and Dickey, 1953).

Chemical changes such as those cited in consumer complaints of dye fading on cellulose acetate, cotton and rayon, can take place within three months at NO_2 concentrations of 380 $\mu g/m^3$ (0.2 ppm) (Hemphill, 1976). Also, the additional acid introduced by SO_2, frequently present in significant concentrations in ambient air, appears to accelerate the fading by NO_2 even though SO_2, by itself, produces no change. (See Table 13-1.)

13.1.1.2 Fading of Dyes on Cotton and Viscose Rayon (Cellulosics)--Although the effects of NO_x on dyed acetate are well documented, the effects on dyes used for the cellulosic fibers have received much less attention. Anomalous cases of fading were reported by McLendon and Richardson (1965) in their study of color changes of dyed cotton placed in gas-heated clothes dryers. Additional effects reported by other investigators are summarized in Table 13-1.

The American Association of Textile Chemists and Colorists (AATCC) conducted service exposure trials to determine whether air contaminants could be one of the variables in lightfastness tests (Salvin, 1964; Schmitt, 1960). Urban and rural sites were chosen in areas of high and low atmospheric contaminant concentrations: Phoenix, Arizona (low), Sarasota, Florida (low), Los Angeles, California (high), and Chicago, Illinois (high). Sulfur dioxide, oxides of nitrogen and O_3 were monitored in each exposure area. A wide range of fibers was dyed with a range of dyes in common use on the fibers (Table 13-2). The fabric samples were exposed to ambient air for 30 and 120 days in covered cabinets which excluded the action of light. Fading was demonstrated on a range of fabrics, including cotton and rayon, for which the cause could be NO_x, O_3 or SO_2. The dyes applicable to cellulosics which exhibited appreciable color change represented four major classes: direct, vat, sulfur and fiber-reactive dyes.

Table 13-3 presents typical atmospheric pollutant concentrations in Los Angeles, Chicago and the rural exposure sites. Chicago's high SO_2 concentration was principally due to burning of coal; ozone concentrations were low. The concentrations of NO_x are high in both Los

TABLE 13-1. FADING OF DYES ON CELLULOSE ACETATE AND CELLULOSICS (COTTON AND RAYON)

			Concentration of Pollutant				
Dyed Fiber	Exposure	Pollutant	μg/m^3	ppm	Time	Effect	Reference
Acetate	Gas heated rooms	NO_2	3,760	2.0	N/A	Fading	Rowe and Chamberlain, 1937
Acetate	Chamber	NO_2	3,760	2.0	16 hr	Fading	Seibert, 1940
Acetate	Pittsburgh-Urban, Ames-Rural	NO_2-O_3	N/A		6 mo	Fading	Salvin and Walker, 1955
Acetate	Chamber	NO_2	3,760	2.0	16 hr	Fading	Salvin, et al., 1952
Cotton-Rayon	Clothes dryer	NO_2	1,128-3,760	0.6-2.0	1 hr cycle	Fading	McLendon and Richardson, 1965
Acetate-Cotton, Rayon	Los Angeles[a]	NO_2	489	0.26	30 to	Fading	Salvin, 1964
		+ O_3	412	0.21	120		
		+ SO_2	131	0.05	days		
	Chicago[a]	+ NO2	414	0.22			
		+ O_3	10	0.005			
		+ SO_2	655	0.25			

(continued)

TABLE 13-1 (continued)

Dyed Fiber	Exposure	Pollutant	Concentration of Pollutant µg/m^3	ppm	Time	Effect	Reference
Cotton-Rayon	Chamber	SO_2-NO_2	3,760	2.0	16 hr	Fading	Salvin, 1969
Cotton-Rayon	Chamber	SO_2-NO_2 and 03	N/A		54 hr	Fading	Ajax et al., 1967
Range of Fibers	Field-Urban, Rural	SO_2-NO_2+O_3	N/A		24 mo	Fading	Beloin, 1972
Range of Fibers	Chamber	NO_2	94 to 940	0.05 to 0.5	12 wk	Fading	Beloin, 1973
Range of Fibers	Chamber	NO_2 + Xenon arc radiation	940	0.5	20 to 80 hr	Fading	Hemphill et al., 1976
Acetate-Cotton, Rayon	Chamber	NO_2	94 to 940	0.05 to 0.5	N/A	Fading	Upham et al., 1976
Acetate-Cotton, Rayon	Survey	NO_2, SO_2, H_2S	Service Complaints		N/A	Fading	Upham and Salvin, 1975

[a] Concentrations also shown in Table 13-3

TABLE 13-2. COLOR CHANGES ON DYED FABRIC--EXPOSED WITHOUT SUNLIGHT IN POLLUTION AND RURAL AREAS

International Grey Scale;[a] 5 = no change; Y = yellow; W = weaker; G = greener; R = redder; and B = bluer

Code Index No.	Phoenix	Los Angeles	Chicago	Sarasota
ACETATE				
Disperse Red 35	4.5Y	4.0Y	4.5Y	4.5Y
Disperse Blue 27	3.0W	2.0W	2.5W	2.0W
Oxides of nitrogen fading control				
Disperse Blue 3	3.5	1.5R	2.0R	3.5
Ozone control--grey dyed with: Disperse Blue 27 Disperse Red 35 Disperse Yellow 37	3.0	1.5	3.5	2.5
POLYESTER				
Disperse Yellow 37	4.5	5.0	4.0	4.0
Disperse Blue 27	4.5	4.0	3.5	4.5
Disperse Red 60	5.0	5.0	4.0	4.5
WOOL				
Acid Black 26A	5.0	4.5	3.5	4.5
Acid Red 89	5.0	3.5	3.5	2.5Y
Acid Violet 1	4.5	4.0	3.0	4.0
Acid Blue 92	4.5	4.0	2.5	4.0
Acid Red 18	5.0	4.0	3.5	4.5
COTTON				
Direct Dyes				
Direct Red 1	4.0	1.5	1.5	3.0
Congo Red B	4.0	2.5	2.5	2Y
Direct Red 10	4.5	3.5	3B	4.0
Direct Blue 76	4.0	2 grey	1R	4.0
Direct Blue 71	4.0	2.5R	2R	3R
Direct Blue 86	4.0	1G	1G	2.5G
Vats				
Vat Yellow 2	5.0	4.0	3G	5.0
Vat Blue 29	3G	3G	2.5G	1.5G
Vat Blue 6	4.0	3.5R	3.0	4R
Vat Red 10	5.0	4.5	3.5	5.0

(continued)

SOURCE: Salvin, 1964.

TABLE 13-2 (continued)

Code Index No.	Phoenix	Los Angeles	Chicago	Sarasota
Fiber Reactives				
Reactive Yellow 4	5.0	5.0	4.5	4.5
Reactive Red 11	5.0	5.0	4.5	5.0
Reactive Blue 9	4.5	4R	3.0R	4.5
Reactive Yellow 16	5.0	5.0	4.0	5.0
Reactive Yellow 13	5.0	5.0	4.0	5.0
Reactive Red 23	5.0	5.0	4.5	5.0
Reactive Red 21	5.0	5.0	4.0	5.0
Reactive Blue 19	4.5	3.0	1R	4.0
Reactive Blue 21	4G	1.5G	1.5G	3G
Reactive Yellow 12	5.0	4.5	3.5	4.5
Reactive Red 19	5.0	5.0	3.5B	4.5
Reactive Red 20	5.0	4.0	4.0	4.5
Reactive Blue 17	4.5	3.0	1 grey	4.0
COTTON				
Sulfur Dyes				
Sulfur Yellow 2	3.5R	3R	2.0R	2.5R
Sulfur Brown 37	5.0	4.5	4.5	4.0
Sulfur Green 2	3B	2.0B	2.0B	2B
Sulfur Blue 8	4.0	3.5G	3G	4.0
Sulfur Black 1	4.5	4.5	4.5	4.5
NYLON				
Acid Red 85	5.0	4.5	3.0	5.0
Acid Orange 49	5.0	4.5	3.0	4.5
Disperse Blue 3	5.0	4.0	3.5	3.0W
Disperse Red 55	5.0	4.5	4.0	3.5
Disperse Red 1	5.0	4.5	3.0	4.5
Alizarine Light Blue C	5.0	4.5	3.5	4.5
ORLON				
Basic Yellow 11	5.0	4.0	4.0	4.5
Basic Red 14	5.0	5.0	4.5	4.5
Basic Blue 21	4.5	4.5	4.0	5.0
Disperse Yellow 3	4.0	5.0	4.5	4.5
Disperse Red 59	5.0	5.0	4.5	4.5
Disperse Blue 3	5.0	5.0	4.0	4.5

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[a] The International Grey Scale is a numerical method of showing the degree of shade change. It is geometric rather than arithmetic. Essentially, a shade change of 4 shows a change which is slight and is not too easily recognized. A shade change of 3 is appreciable and is easily recognized. A shade change of 2 is severe. A shade change of 1 is disastrous. These numbers are indicative of the shade change with 4 being passable and 3.5 a matter of judgement.

TABLE 13-3. TYPICAL CONCENTRATIONS[a] OF ATMOSPHERIC CONTAMINANTS IN EXPOSURE AREAS

	Rural (Phoenix - Sarasota)		Los Angeles		Chicago	
	(ppm)	($\mu g/m^3$)	(ppm)	($\mu g/m^3$)	(ppm)	($\mu g/m^3$)
Oxides of Nitrogen	0.01		0.26		0.22	
Sulfur Dioxide	0.03	80	0.05	130	0.25	650
Carbon Monoxide	--		23.00	26,000	16.00	18,000
Ozone	0.06-0.11	120-220	0.21	410	0.005	10
Aldehydes	--		0.3			

[a] Concentrations shown are average concentrations measured over a two-month period, relative humidity data not available.

SOURCE: Salvin, 1964.

Angeles and Chicago, and are of similar magnitude. These differences in pollutant concentrations correlate with the fact that the Disperse Blue 3, which characteristically reacts to NO_x, showed pronounced reddening changes in both Los Angeles and Chicago while being almost unchanged (in the International Grey Scale ratings) in the rural exposure areas of Phoenix and Sarasota (Table 13-2). It should be noted that humidity differences are present between Phoenix, which is dry, and coastal Florida, which is humid. Humidity was not measured in Los Angeles and Chicago.

In a laboratory experiment (Salvin, 1969) designed to produce changes similar to those shown on service exposure, the AATCC NO_x test with Disperse Blue 3 was not used. Instead, the German Fastness Commission test was used, which has shown changes in dyes on cellulose. In this method, discussed by Rabe and Dietrich (1956) oxides of nitrogen are generated by the addition of phosphoric acid to a dilute sodium nitrite solution. The dyed fabric is exposed to the nitrogen oxides in a closed system under high humidity conditions, in contrast to the AATCC test procedure, in which the nitrogen oxides are generated by combustion of natural gas or butane under conditions of low humidity. The dyed fabrics which showed fading changes in the service exposures in Los Angeles and Chicago showed similar changes upon laboratory exposure under the high humidity conditions of the German Fastness Commission test procedure (Salvin, 1969).

The effect of NO_x on fiber reactive dyes has been reported by Imperial Chemical Industries in its shade card of Procion Dyes. The vulnerability of certain reactive dyes on cotton to NO_x also has been reported by Hertig (1968) in his critical study of the International Standards Organization test procedure (Rabe and Dietrich, 1956) for color fastness to NO_x. This method employs high humidity conditions.

The effects of air pollutants were examined by the U.S. Environmental Protection Agency (USEPA) in laboratory trials (Ajax et al., 1967) using the same dye-fabric combinations employed in the AATCC (Salvin, 1964) study (Table 13-2). The dye-fabric combinations were exposed to air to which diluted auto exhaust and SO_2 were added over a 54-hour period. Neither the auto exhaust nor SO_2 produced significant fading. However, irradiation of the auto exhaust, which contains both hydrocarbons and oxides of nitrogen, gave products which caused significant fading. The addition of SO_2 at a concentration of 2,620 $\mu g/m^3$ (1.0 ppm) produced additional fading. The synergistic effect of SO_2 is suggested as being responsible for the observed results.

Beloin (1972) carried out a USEPA field exposure study of the fading of 67 dye-fabric combinations (using 56 dyes) representative of the AATCC service exposure described in Table 13-2. The exposures were carried out in eleven nationwide urban and rural sites for consecutive three-month periods over a period of two years.

The exposure sites are listed in Table 13-4. Rural areas were selected to serve as controls with the same climatic conditions as the urban areas but with low levels of pollution. Phoenix, Arizona and Sarasota, Florida were chosen as low pollution areas with extremes in

TABLE 13-4. EXPOSURE SITES

City	Location	Type	Average Fade, NBS Units[a]
Washington, DC	Municipal Building	Urban	5.0
Poolesville, MD	Poolesville High School	Rural	4.3
Tacoma, WA	Franklin Gault School	Urban	4.3
Purdy, WA	PHS Shellfish Laboratory	Rural	2.9
Los Angeles, CA	LA County Air Pollution Control District Building	Urban	5.7
Santa Paula, CA	Federal Post Office	Rural	4.0
Chicago, IL	Central Office Building	Urban	7.2
Argonne, IL	Argonne National Laboratory	Rural	4.0
Phoenix, AZ	Desert Sunshine Exposure Tests	Suburban	2.7
Sarasota, FL	Sun Test Unlimited	Rural	3.1
Cincinnati, OH	Taft High School	Urban	4.8

[a] On the NBS scale, a change of three units is noticeable; three to six units are considered appreciable; changes above six units are classified as severe.

SOURCE: Beloin, 1972.

relative humidity and high temperatures. The exposures were carried out in louvered covered cabinets to avoid exposure to light. The dye production sales for 28 of the 56 different dyes tested totalled over 30 million dollars (U.S. Tariff Commision, 1967). Of the 67 dye-fabric combinations, 25 were cellulosics.

The sites were monitored for O_3, NO_2 and SO_2. The gas fading control (Disperse Blue 3 on cellulose acetate) showed high correlation with NO_x concentration. Fadings on the O_3 test ribbon were demonstrated in the rural areas as well as the urban sites.

It can be concluded from the data that appreciable fading takes place in the absence of light. Of the 67 dye-fabric combinations tested, 64 percent showed appreciable fading. Urban sites produce significantly higher fading than corresponding rural sites. In the presence of high temperature and high humidity, air pollution increases fading rate. Fading changes were severe in those fabrics dyed with direct and reactive dyes.

The data did not isolate the action of SO_2, O_3, or NO_2, but a statistical analysis identified NO_2 as a significant variable (at 99 percent level) causing fading in some samples. Chicago samples incurred highest average fading. However, Chicago had high concentrations of SO_2, as well as NO_2.

Beloin (1973) also conducted laboratory studies designed to determine the effects of the individual air pollutants on dyed fabrics. Exposure was made of 20 dyed fabrics chosen on the basis of their appreciable change in the field study noted above. Cotton and viscose rayon were 9 of the 20 dyed fabrics (Table 13-5) used in the trials. Reactive dyes and one type of vat dye were used. The pollutants were SO_2, NO, NO_2, and O_3. Two concentration levels were used for nitrogen dioxide: 940 and 94 $\mu g/m^3$ (0.5 ppm and 0.05 ppm). Temperature and humidity were varied: 32°C and 13°C, and 90 and 50 percent relative humidity, respectively. The fabrics were exposed for 12 weeks.

Under the higher humidity/temperature conditions, NO_2 at a concentration of 940 $\mu g/m^3$ (0.5 ppm), caused severe changes on 8 of the 9 samples. Significant fading also occurred at high humidity with an NO_2 concentration of 94 $\mu g/m^3$ (0.05 ppm) (Beloin, 1973). Color changes were reported as Hunter Color Units, which approximate the NBS units.

The AATCC Committee on Color Fastness to Light carried out light-fastness tests with added contaminants using Xenon arc irradiation (Hemphill et al., 1976). The objective was to establish a relationship between light-fastness tests made in natural daylight in Florida and Xenon arc exposure in the laboratory Weatherometer. The contaminants included separate additions of 940 $\mu g/m^3$ (0.5 ppm) NO_2, 294 $\mu g/m^3$ (0.15 ppm) O_3 and 786 $\mu g/m^3$ (0.3 ppm) SO_2. In one trial, all three contaminants were added to the exposure cycle under Xenon arc irradiation. Of the 29 dyed fabric combinations exposed, 14 were cellulosic (cotton or rayon). The addition of NO_2 alone caused increased fading compared to the control in over half of dyed cellulosic fabrics examined.

Upham and co-workers (1976) carried out a chamber study of the effect of atmospheric pollutants on selected drapery fabrics. Fabrics were exposed to 0.05 and 0.5 ppm each of SO_2

TABLE 13-5. AVERAGE FADING OF 20 DYE-FADRIC COMBINATIONS[a] AFTER 12 WEEKS EXPOSURE TO NITROGEN DIOXIDE
Hunter Color Units[b]

			94 µg/m³ NO_2				940 µg/m³ NO_2			
Material	Dye	Color Index No.	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)
Cotton	Direct	Red 1	7.2	8.0	7.4	7.8	18.0	20.4	16.1	22.3
Rayon	Direct	Red 1	3.4	T	T	T	13.4	16.3	12.6	17.0
Wool	Acid	Red 151	T	T	T	T	T	T	T	T
Cotton	Reactive	Red 2	T	T	T	T	10.4	6.9	9.7	7.6
Acrylic	Basic	Red 14	T	T	T	T	T	T	T	T
Cotton	Azoic[c]	Red	T	T	T	T	T	T	T	T
Nylon	Acid	Orange 45	5.6	17.0	10.1	9.5	21.5	27.9	24.3	25.1
Wool	Acid	Yellow 65	T	T	T	T	T	T	T	T
Acrylic	Basic	Yellow 11	T	T	T	T	T	T	T	T
Cotton	Sulfur	Green 2	T	3.3	T	T	6.5	6.6	6.1	7.1
Wool	Acid	Violet 1	T	T	T	T	T	T	T	4.1

(continued)

SOURCE: Beloin, 1973.

TABLE 13-5. (continued)

			94 $\mu g/m^3$ NO_2				940 $\mu g/m^3$ NO_2			
Material	Dye	Color Index No.	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)
Cotton	Direct	Blue 86	5.9	9.5	9.4	6.0	14.1	17.2	14.2	17.1
Cellulose Acetate	Disperse	Blue 3	29.0	42.3	37.7	33.6	86.9	75.6	88.0	74.4
Nylon	Disperse	Blue 3	5.5	14.7	5.9	14.2	39.6	45.5	34.0	51.1
Cellulose Acetate	Disperse	Blue 27	6.4	4.9	3.8	7.5	20.5	26.8	17.0	29.6
Polyester	Disperse	Blue 27	T	T	T	T	T	T	T	T
Cotton	Reactive	Blue 1	3.9	13.6	9.6	7.9	31.8	41.7	35.4	38.1
Cotton	Reactive	Blue 2	6.4	10.6	8.2	8.9	30.5	41.6	33.8	38.4
Cotton	Vat	Blue 14	6.3	6.7	3.3	9.7	34.3	30.4	23.4	41.3
Acetate	---[d]	AATCC Ozone Ribbon	T	T	T	T	5.7	11.7	5.7	11.7

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[a] Each average, e.g. the low temperature average, was calculated by averaging the color change of duplicate samples from both the low temperature-low humidity and low temperature-high humidity exposure periods.

[b] T=trace (less than 3 units of fading). The higher the number, the greater the fading. Hunter Color Units approximate the NBS color scale.

[c] Coupling Component 2, Azoic Diazo Component 32.

[d] C.I. Disperse Blue 27, C.I. Disperse Red 35, C.I. Disperse Yellow 37.

(131 and 1310 µg/m^3), O_3 (98 and 980 µg/m^3), and NO_2 (94 and 940 µg/m^3) under Xenon arc irradiation, at various humidities. The effect of NO_2 was pronounced, especially on a vat-dyed drapery fabric. The most noticeable color changes were at 940 µg/m^3 (0.5 ppm) and 90 percent relative humidity.

In summary, the investigations by Beloin both in the field study (1972) and the chamber study (1973) show that, at concentrations of NO_2 present in urban atmospheres, representative dyes for cotton and rayon will suffer serious fading. NO_2 resistant dyes are available; however, limitations of brightness of shade and difficulty of application introduce production problems and the need for greater quality control resulting, generally, in increased costs. The AATCC work confirms the vulnerability to NO_2 of several dyes widely used on cellulosic fibers, especially certain blue dyes.

13.1.1.3 Fading of Dyes on Nylon--The fading of dyed nylon in polluted atmospheres has been noted in exposure trials carried out by the AATCC (Salvin, 1964) and by the U.S. Environmental Protection Agency (Beloin, 1972). However, consumer complaints have been few and have been blamed on light-produced color changes in garments, draperies, or in home furnishings for the nylon fiber normally used in these products. The standard AATCC test procedure for NO_2, which demonstrated the vulnerability of the disperse dyes used on acetate, showed little change when the same dyes were used on nylon (Table 13-6). The problem of fading on nylon became of considerable interest as nylon found use in carpets. The quality of nylon for this end use is estimated at over 500 million pounds, distributed between Nylon 66 and Nylon 6.

Field exposures of a range of dyes on nylon, in areas of high air pollution (NO_2, SO_2 and O_3), resulted in unexpected failures of these dyes (Beloin, 1972; Salvin, 1964). In contrast, the same dyes on polyester showed no changes (Table 13-6). The fading of disperse blue dyes on nylon carpets was shown to be due to O_3 in the presence of high humidity (Salvin, 1974b). Acid dyes, which are more resistant to O_3, were substituted as a remedial measure. However, the remedy presented additional problems. The vulnerability of acid dyes on nylon to NO_2 was the basis of a bulletin issued by Imperial Chemical Industries (1973) on a range of acid dyes marketed as Nylomines. The fading effect of NO_2, derived from combustion gases, was determined at 65 and at 95 percent relative humidity on three cycles of exposure in their tests. Certain violet and blue dyes are rated as exhibiting significant change. The dye manufacturers point out the importance of dye selection in carpets and in home furnishings which are likely to be exposed for long periods in air contaminated with NO_2 from gas burner fumes.

Acid dyes on nylon were included in the range of dyes exposed to visible light radiation and NO_2 by Hemphill in the AATCC study (Hemphill et al., 1976). Under high humidity conditions and at an NO_2 concentration of 940 µg/m^3 (0.5 ppm), for 30 to 100 hours, fading was found to be greater for certain dyes in those exposures where Xenon arc irradiation and NO_2 were present than in the control exposure with Xenon arc irradiation and NO_2-free air (Table 13-6).

13.1.1.4 Fading of Dyes on Polyester--Polyester dyed with disperse dyes did not show NO_2-induced fading changes in AATCC field exposures in urban atmospheres of Chicago and Los Angeles (Table 13-6) (Salvin, 1964). The same fabrics in various urban sites (Beloin, 1972)

TABLE 13-6. EFFECT OF NITROGEN DIOXIDE ON FADING OF DYES ON NYLON AND POLYESTER

Dyed Fibers	Exposure	Pollutant	Concentration of Pollutant		Time	Effect	Reference
			µg/m^3	ppm			
Nylon	Chicago Los Angeles	NO_2	188 282	0.1 0.15	30 to 120 days 30 to 120 days	Fading	Salvin, 1964
Polyester	Chicago Los Angeles	NO_2	376 282	0.2 0.15	30 to 120 days 30 to 120 days	Unchanged	*Ibid*.
Nylon	Urban Sites	NO_2	376	0.2	3 to 24 months	Fading	Beloin, 1972
Polyester	Urban Sites	NO_2	376	0.2	3 to 24 months	Unchanged	*Ibid*.
Nylon	Chamber High Humidity	NO_2	188 to 1,880	0.1 to 1	12 weeks	Fading	Beloin, 1973
Polyester	Chamber High Humidity	NO_2	188 to 1,880	0.1 to 1	12 weeks	Unchanged	*Ibid*.
Nylon	Chamber High Humidity	NO_2	376	0.2	48 hours	Fading	Imperial Chemical Industries, 1973
Nylon	Chamber High Humidity Xenon Arc	NO_2	940	0.5	30 to 120 hours	More fading than without NO_2	Hemphill et al., 1976
Polyester Permanent Press	Chamber	NO_2	940	0.5	16 hours	Fading	Salvin, 1966
Polyester Textured Double Knit	Chamber	NO_2	940	0.5	16 hours	Fading	Urbanik, 1974

also showed no changes; high or low humidity had no effect. However, a large number of complaints regarding fading of permanent press garments (65 percent polyester--35 percent cotton) were recorded in 1965, when this product first was marketed.

Investigation of the anomalous fading of polyester dyes in permanent press fabrics by Salvin (1966) indicated that fading did not take place on dye contained within the polyester fiber matrix. Fading was found to take place on the surface of the fiber as a result of dye migration from the fiber subsurface to the modified urea-formaldehyde resin used on the blend to stabilize the cotton. When exposed to NO_2 or O_3, the components of the resin finish absorb the contaminants and fading occurs. The measures which can be used to eliminate the problem are (a) selection of dyes with lower rate of migration, (b) substitution of the magnesium chloride catalyst with zinc nitrate and, (c) reduction in quantity of non-ionic surfactant which acts as an acceptor for the dye and an absorber for either NO_2 or O_3. Urbanik (1974) suggests changes in the magnesium chloride catalyst formulation, used in the resin finish, in order to suppress dye migration.

The introduction, in 1966, of double-knit garments made from textured polyester also was accompanied by cases of fading of the garments attributed to either NO_2 or O_3. The fading attributed to NO_2 was noted in those dyes of level dyeing properties. Dye migrates in the final heat-setting step to lubricant oils or residual surfactant on the surface of the fabric, where fading takes place.

13.1.1.5 Economic Costs of NO_x -induced Dye Fading--Barrett and Waddell (1973), in a 1973 status report to the U.S. Environmental Protection Agency, reported preliminary estimates that annual economic costs of NO_x-induced dye fading in textiles amounted to $122.1 million. These estimates were based on figures reported by Upham and Salvin (1975).

The economic costs to the nation as a result of O_3 damage to textiles is approximately 70 percent of the costs attributed to NO_x damage. These costs of NO_x and O_3 action are tabulated in Table 13-7.

The basis for the estimates included not only the reduced wear life of textiles of moderate fastness to NO_x but also the costs of research and quality control. The major share of the costs is the extra expense involved in using dyes of higher NO_x resistance and in the use of inhibitors. Additional costs also are incurred in dye application and in increased labor expenditures.

The factors relating to higher costs in the textile industry as a result of NO_x action are discussed in Chapter 8, "The Effects of Nitrogen Oxides on Materials," in the National Academy of Sciences report on nitrogen oxides (National Academy of Sciences, 1976a).

13.1.2 Yellowing of White Fabrics by NO_2

The survey of the effects of air pollutants on textiles (Upham and Salvin, 1975) reported a number of instances in which white fabrics yellowed. This discoloration occurred in areas protected from light. Causes of yellowing were not established, except for the observation that contact with ambient air currents seemed to be a causative factor.

TABLE 13-7. ESTIMATED COSTS OF DYE FADING IN TEXTILES

Pollutant	Effect	\$ million[a]
NO_x	Fading on acetate and triacetate	73
	Fading on viscose rayon	22
	Fading on cotton	22
	Yellowing of white acetate-nylon-Spandex	6
	Subtotal	122
O_3	Fading on acetate and triacetate	25
	Fading on nylon carpets	42
	Fading on permanent-press garments	17
	Subtotal	84
	Total	206

[a] All costs rounded to nearest million, therefore some totals do not agree.

SOURCE: Barrett and Waddell, 1973.

Using 18 fabric samples which were the subjects of manufacturers' complaints, Salvin (1974c) investigated the effects of specific air pollutants or combinations and the effects of humidity and temperature. The products tested include polyurethane segmented fiber, rubberized cotton, optically-brightened acetate, nylon, nylon treated with permanent antistatics, and resin-treated cotton containing softeners (Table 13-8).

Nitrogen dioxide was established as the pollutant responsible for yellowing of white fabrics in the complaint fabrics tested. Yellowing was not demonstrated when fabrics were exposed to O_3, SO_2, or hydrogen sulfide (H_2S).

The standard AATCC test procedure (conducted in low humidity) for effects of NO_2 does not always result in the yellowing effect observed on service exposure in areas shielded from light. This is especially true of cotton and nylon fabrics. Whereas the standard test procedure for NO_x showed change on cellulose acetate fabrics, high humidity test procedures demonstrated yellowing in nylon.

Although fibers without additives do not show yellowing following exposure to NO_2, the polyurethane-segmented fibers (e.g., Lycra and Spandex) are exceptions. These fibers contain urethane groups which react directly with NO_2 to form yellow-colored nitroso compounds. Yellowing of Spandex and Lycra fibers occurred in the standard AATCC test for NO_2 fading (Salvin, 1974c). In the tests carried out, less yellowing was shown on certain samples submitted which contained inhibitors.

Garments containing rubberized cotton did show yellowing on exposure to NO_2 in the AATCC standard test procedure. With increase in temperature of the test, yellowing was more pronounced in those areas of the garment where rubber is present (Burr and Lannefeld, 1974).

The antioxidant in rubber employed against ozone action is diphenyl ethylene diamine. On storage, this product vaporizes from the rubber to the surface of the cotton fabric. This material already is well known as the inhibitor used on cellulose acetates to suppress fading of dyes by NO_2. It forms a yellow nitroso compound.

Optical brighteners, compounds widely used to improve the whiteness of fabrics, are of various structures and are specific to particular fibers. They function by transforming UV radiation to visible light in the purple-blue range. Brighteners may react with NO_2 and result in yellow-colored compounds.

The AATCC (1957) has conducted a study of the yellowing of a range of softeners. In the range of fabrics examined for yellowing of the softeners, variable degrees of yellowing were demonstrated upon exposure to NO_2. The yellowing was especially noted on those softeners of a cationic nature. Softeners have been synthesized which are resistant to yellowing by oxides of nitrogen (Dexter Chemical Company).

The unexpected yellowing of nylon treated with an antistatic agent has been reported (Salvin, 1974c). The treated nylon showed no yellowing on exposure in the standard NO_2 AATCC test method. Under high humidity and with an increase in temperature during testing, however, yellowing similar to that obtained in service testing was observed.

TABLE 13-8. YELLOWING OF WHITES BY NITROGEN DIOXIDE

Fiber	Exposure	Pollutant	Concentration of Pollutant $\mu g/m^3$	ppm	Time	Effect	Reference
Survey	Service Complaints	N/A			N/A	Yellowing	Upham and Salvin, 1975
Rubberized Cotton	Chamber	NO_2	376	0.2	16 hr	Yellowing	Burr and Lannefeld, 1974
Rubberized Cotton	Chamber	NO_2	376	0.2	16 hr	Yellowing of anti-oxidant	Salvin, 1974c
Spandex	Chamber	NO_2	376	0.2	8 hr	Action on fiber	Ibid.
Acetate Optical brightener	Chamber	NO_2	376	0.2	8 hr	Yellowing	Ibid.
Nylon Optical brightener	Chamber High Humidity	NO_2	376	0.2	16 hr	Yellowing	Ibid.
Nylon Anti-stat finish	Chamber High Humidity	NO_2	376	0.2	16 hr	Yellowing	Ibid.
Cotton Cationic softener	Chamber	NO_2	376	0.2	16 hr	Yellowing	Ibid.

13.1.3 Degradation of Textile Fibers by Nitrogen Oxides

Cotton and nylon are the two fibers whose strength is reduced by the hydrolytic action of acid aerosols. This problem assumes economic importance because industrial fabrics comprise the end use for 18 percent of all fibers, many of which are used in the production of cordage, belts, tarpaulins and awnings. These products are exposed to air pollutants over long periods of time. Premature losses of strength are costly and create safety hazards.

A chamber study of the combined action of NO_2 and light on cotton did demonstrate that NO_2 contributed to strength loss (Morris, 1966). Cotton yarns were exposed to sunlight. In one cabinet, air was filtered through carbon to remove oxides of nitrogen. The second cabinet contained fibers exposed to sunlight and air containing monitored levels of O_3 and NO_2. Exposure was carried out for a total of 72 days. Since the test area (Berkeley, California) was low in SO_2, this service exposure would emphasize the effects of nitrogen oxides. The degradation (strength loss) of the cotton fiber was greater in the presence of the unfiltered ambient air. The results of this study show that the combined effects of sunlight and NO_x gave increased deterioration over sunlight alone. The contributions of O_3 and other oxidants was not determined.

It is not possible to isolate the effects of nitrogen oxides from that of sulfur oxides in field studies which have shown strength loss in cotton (Brysson et al., 1967; Brysson et al., 1968; Morris, 1966; Travnicek, 1966). The effect of NO_2 on fiber degradation of cotton requires further investigation.

Inconclusive results were shown by Zeronian et al. (1971) in a study of the effect of NO_2, combined with Xenon arc radiation, on a range of man-made fibers, including acrylics, nylons and polyester. These workers exposed modacrylic-acrylic, nylon, and polyester fibers to the combined action of Xenon arc radiation and air containing 376 $\mu g/m^3$ (0.2 ppm) NO_2 at 30 percent relative humidity at 48°C, and at 43 percent relative humidity at 43°C. The fabrics were exposed for 108 hours to Xenon arc irradiation in a Weatherometer and intermittently were sprayed with water. The same series of fabrics was subjected to identical weathering conditions but without NO_2. Modacrylic (Dynel), acrylic (Orlon), and polyester showed only slight differences in degradation in exposures with and without NO_2. These materials are considered resistant to the action of acids. The results for nylon were not conclusive, although significantly greater degradation (loss of tensile strength, increased viscosity) at 48°C occurred in the presence of NO_2, under irradiation. Further experimentation would be suggested to test NO_2 effects in the absence of irradiation and under higher humidity conditions.

13.2 EFFECTS OF NITROGEN DIOXIDE ON PLASTICS AND ELASTOMERS

The maintenance of strength of plastics and elastomers upon exposure of these materials to light, air and atmospheric contaminants is a matter of importance.

A 1977 survey (Chemical and Engineering News, 1977) predicts a market for these materials in 1982 of 1.78 billion pounds. Under the generic term of plastics are included polyethylene, propylene, polystyrene, polyvinyl chloride, polyacrylonitrile and polyamides.

Ageing is the term used to denote deterioration of chemical and physical properties which can occur upon weathering. This degradation has been blamed on the known effects of sunlight. The comparative resistance to sunlight can be shown upon exposure in the laboratory to carbon or Xenon arc irradiation. The substitution of plastics for metals, particularly in automobiles, where long periods of outside exposure are involved, brings this problem into focus. The effects of air pollutants, such as NO_2, have been assessed in laboratory trials.

The polymers which represent the structures in plastics, as well as textiles, were subjected by Jellinek et al. (1969) to the action of SO_2, NO_2, and ozone obtained by action of UV radiation on the oxygen-containing mixture. These combinations of SO_2, NO_2, and ozone represent the components of smog. The polymers include polyethylene, polypropylene, polystyrene, polyvinyl chloride, polyacrylonitrile, butyl rubber and nylon. All polymers suffered deterioration in strength. The elastomer, butyl rubber, was more susceptible to SO_2 and NO_2 than other polymers. However, the effect of O_3 on the rubber was more pronounced than that of the SO_2 or the NO_2.

Jellinek (1970) examined the reaction of linear polymers, including nylon and polypropylene, to NO_2 at concentrations of 1,880 to 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm). Nylon 66 suffered chain scission. (Chain scission results in polymers having lower molecular weights and lower strength.) Polypropylene tended to crosslink. Chain scission of polymers caused by small concentrations of SO_2 and NO_2 took place in the presence of air and UV radiation.

The action of NO_2 and O_3 on polyurethane also was investigated by Jellinek (1974). The tensile strength of linear polyurethane was reduced by NO_2 alone and also by NO_2 plus O_3. Chain scission resulting in lower molecular weights and formation of nitro- and nitroso-groups along the polymer backbone occurred upon exposure to NO_2.

13.3 CORROSION OF METALS BY NITROGEN DIOXIDE

The corrosion of metals by air pollutants is due to the presence of acids or salts on the metal which enter into electrochemical reactions. Numerous small electrochemical cells form on the ferrous metal surfaces which are in contact with the contaminated air. Localized anodes and cathodes form and the electrical conductivity is increased where the surface of the metal is wet; the moisture contains increasing quantities of acid aerosols or anions therefrom.

The normal rusting process is the formation of an iron-oxide which acts as a protective film. However, the presence of an acid aerosol such as sulfuric acid, as derived from SO_2, can break down this protective oxide layer, exposing new surfaces to electrolytic corrosion.

An analysis of the contributing effects of air pollutants was offered in the examination of the contributions of NO_2 to corrosion in the review by the National Research Council (National Academy of Sciences, 1976b).

The forms of metal corrosion include new corrosion (uniform and general attack), galvanic corrosion, crevice corrosion, pitting, selective leaching, and stress corrosion. A liquid film or the presence of a hydrated salt plays a role in most of these corrosion types. In attempting to assess the contribution of nitric acid aerosols derived from NO_2 and nitrate salts, the various mechanisms of corrosion must be understood to attenuate the problem and to predict the effect of a variable change.

Thus the mechanisms of galvanic, pitting, crevice, and selective leaching corrosion require the presence of an electrolyte or solvent. Hydrated salt solutions can serve as the electrolyte in galvanic corrosion.

The general approach taken by investigators to the effects of air pollutants on corrosion has been to establish the extent of the damage with the goal of obtaining data which could be used in economic analyses of corrosion due to air pollutants. Waddell (1974)in his estimates of the economic damages of air pollution includes a section on costs of metal corrosion. Using data supplied by the Rustoleum Corporation, Barrett and Waddell (1973) estimated costs at $7.5 billion in 1958.

Material damage due to air pollutants emphasizes SO_2 as the major causative agent in the corrosion of metals (Fink et al., 1971; Gillette, 1975). Fink and co-workers (1971) estimated corrosion damage of metals caused by air pollution at approximately $1.5 billion.

When consideration is given to the mechanisms of corrosion which are due to electrolytic action rather than oxidation, the importance of NO_2 in the corrosion process emerges. The presence of nitrates on surfaces, to give both the requisite hygroscopic film and the electrolyte for galvanic corrosion, becomes important. The potential of nitrates in upsetting the homogeneity of the protective oxide film becomes a factor.

Haynie et al. (1976) investigated the separate or combined effects of SO_2, NO_2 and O_3 under controlled conditions of pollutant, humidity and temperature. The materials tested included: weathering steel, galvanized steel, aluminum alloys, paints, drapery fabrics, vinyl house siding, marble, and white sidewall tire rubber. Sulfur dioxide and relative humidity appeared the most important factors for producing corrosion. A similar conclusion was reported by Yocum and Grappore (1976)in a review of the effect of air pollutants emanating from power plants.

The premature failure of nickel brass springs in telephone equipment primarily in the Los Angeles area has been investigated by Hermance and co-workers (1971). Although most failures occurred in California, springs did show occasional failure in other parts of the country. This report reviewed the earlier findings that the springs had a fogged appearance and were covered with a hygroscopic dust rich in nitrates. The failure was attributed to stress corrosion. A survey of nitrate accumulation was made in California and other locations. Nitrate deposition correlated with relay failure.

Previous laboratory studies by Hermance (1966) and McKinney and Hermance (1967) confirmed that hygroscopic nitrates such as zinc or ammonium cause stress corrosion cracking of the anodic nickel brass wires. Salts of other anions under the same conditions did not cause cracking at the nitrate concentration levels found in Los Angeles. Up to about 15 $\mu g/cm^2$ of surface area, an applied positive potential was necessary. Cracking was found to be low when the relative humidity was less than 50 percent.

A field study was made of the incidence of breakage as related to the nitrate accumulation. The nitrate accumulations were measured in New York City, Bayonne, New Jersey, Philadelphia, Baltimore, and Washington, D.C. Although the accumulations were high, breakage in these areas was lower than anticipated.

The important finding illustrated in this work is that the nitrates salts are more hygroscopic than the chloride and sulfate salts. As such, they may lower the threshold humidity requirements for the formation of an aqueous medium, which can serve as the electrolyte or solvent for wet corrosion.

Hermance (1966) also reported on the failure of other telephone equipment, which did not involve nickel brass alloys, that took place in Los Angeles, New York, Detroit and Cleveland. The nickel base of palladium-capped contacts of cross box switches corroded in the presence of nitrates, forming bright greenish corrosion products which gradually crept over the palladium cap of the contacts. The heavy nitrate deposits were greater than 15.5 $\mu g/cm^2$. Stress corrosion occurred in the absence of anodic electrical current.

The function of NO_2 in changing the defect structure of many oxides, thereby increasing or decreasing the rate of oxidation of metals and alloys, was suggested by Lazareva et al. (1973) in the study of the oxidation of tungsten alloys.

A field study of the effect of air pollutants on electrical contact materials was carried out by Chiaranzelli and Joba (1966), in which the formation of corrosion films in various areas of pollution was correlated with the concentration of pollutants present. Nitrogen dioxide, SO_2, H_2S and dust were monitored. This study did not isolate the specific contribution of NO_2 to the problem of electrical contacts although it did point out that areas of high humidity showed greater corrosion.

It is in the study of catastrophic failure of materials exposed to air pollutants that investigators seek to establish the specific causative agent. The long-term exposures by the ASTM of various metals in different locations sought to establish which metal or alloy was most resistant. The case of the telephone equipment failure as investigated by Hermance et al (1971) did show that nitrates were a contributory factor, although no relation to the concentration of NO_2 in the air was established.

Gerhard and Haynie (1974) examined the cases of catastrophic failure of metals in which structures failed unexpectedly, leading to loss of life as well as well as collapse of the metal structure. Their conclusion was that air pollutants were a probable contribution to the corrosion that was the cause of failure. However, there is no finding that determined the relationship between levels of particular pollutants and the occurrence of the failure.

Nitrogenous compounds, however, were implicated in a situation in which steel cables on a bridge in Portsmouth, Ohio failed after 12 years of service. The cause of failure was traced to river water contaminated with ammonium nitrate that had concentrated at natural crevices ([illegible], 1965). Nitrogen dioxide was not considered a factor.

A review of the voluminous literature on corrosion has produced no further references to investigations of NO_2 action, in the absence of SO_2. The above studies are summarized in Table 13-9.

13.4 SUMMARY

The damaging effects of atmospheric oxides of nitrogen have been established for a variety of materials including dyes, fibers, plastics, rubber and metals. Field exposures of cotton, viscose rayon, cellulose acetate, and nylon fabrics colored with representative dyes demonstrate that fading occurs for specific dyes in air containing NO_2, O_3, and SO_2. These exposures were carried out in ambient air and protected against sunlight. Chamber studies using individual pollutants NO_2, O_3, and SO_2 have shown that some individual dye-fiber combinations exhibit color fading only in response to NO_2 exposure, whereas others are susceptible to O_3, as well as combinations of NO_2 and O_3. SO_2 introduced an accelerant effect. Disperse dyes used for cellulose acetate and rayon include vulnerable anthraquinone blues and reds. The cellulosic fibers cotton and viscose rayon, dyed with certain widely used direct dyes, vat dyes, and fiber reactive dyes, suffer severe fading on chamber exposures to 940 $\mu g/m^3$ (0.5 ppm) NO_2 under high humidity (90 percent) and high temperature (90^oF) conditions. Significant fading is observed on 12 weeks exposure to 94 $\mu g/m^3$ (0.05 ppm) NO_2 under high humidity and temperature conditions (90 percent, 90^oF).

Acid dyes on nylon fade on exposure to NO_2 at levels as low as 188 $\mu g/m^3$ (0.1 ppm), under similar conditions. Dyed polyester fabrics are highly resistant to NO_2-induced fading. However, permanent press fabrics of polyester cotton and textured polyester exhibited unexpected fading when first marketed. The fading was in the disperse dye which migrated under high heat conditions of curing or heat setting to the reactive medium of resins and other surface additives.

The yellowing of white fabrics is documented for polyurethane segmented fibers (Lycra and Spandex), rubberized cotton, optically brightened acetate, and nylon. Yellowing is also reported on fabrics which were finished with softeners or anti-static agents. Nitrogen dioxide was demonstrated to be the pollutant responsible for color change, with O_3 and SO_2 showing no effect. Chamber studies using NO_2 concentrations of 376 $\mu g/m^3$ (0.2 ppm) for 8 hours showed yellowing equivalent to that on garments returned to manufacturers.

The tensile strength of fabrics may be adversely affected by the hydrolytic action of acid aerosols. Nitrogen dioxide has been demonstrated to oxidize the terminal amine group ($-NH_2$) of nylon to the degree that the fiber has less affinity for acid-type dyes. Nylon 66 may suffer chain scission when exposed to 1,880 to 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm) NO_2. Field exposures of fibers emphasize the action of acids derived from SO_2, although NO_2 may also be present in high concentrations in urban sites. Information on the contribution of NO_2 to degradation is incomplete.

TABLE 13-9. CORROSION OF METALS BY NITROGEN DIOXIDE

Metal	Exposure	Pollutant	Effect	Reference
Mechanics of Corrosion - Function of Nitrates				National Academy of Sciences, 1976a
Nickel Brass	Los Angeles	Nitrates	Strength Loss	Hermance et al., 1971
Nickel Brass	Los Angeles	Nitrates	Strength Loss	McKinney and Hermance,1967
Nickel	Los Angeles New York	Nitrates	Corrosion	Hermance, 1966
Tungsten	Chamber	NO_2	Change oxide surface	Lazareva, 1973
Electronic contacts	Field	NO_2-SO_2-H_2S	Corrosion film	Chiaranzelli and Joba, 1966
Metal parts	Field	NO_2-SO_2-O_3	Failure	Gerhard and Haynie, 1974
Economic Costs of Corrosion				Fink et al., 1971

Although a survey of the market for plastics predicts the use of 1.78 billion pounds in 1982, there is essentially no information on the effects of NO_2 on polyethylene, polypropylene, polystyrene, polyvinyl chloride, polyacrylonitrile, polyamides and polyurethanes. Aging tests involve sunlight exposure as well as exposure to ambient air. Chamber exposure of the above plastics to combinations of SO_2, NO_2, and O_3 has resulted in deterioration. Nitrogen dioxide alone has caused chain scission in nylon and polyurethane at concentrations of 1,880 and 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm).

The extensive data on corrosion of metals in polluted areas relate the corrosion effects to the SO_2 concentrations. The presence of NO_2 and its contribution is not evaluated despite its presence as acid aerosol in appreciable concentrations.

Ammonium nitrates were implicated as a factor in the stress corrosion cracking of wires made of nickel brass alloy used in telephone equipment. Since nitrate salts have been shown to be more hygroscopic than either chloride or sulfate salts, the presence of nitrates may lower the humidity requirements for the formation of an aqueous electrolyte system in the wet corrosion of metals.

CHAPTER 13

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14. STUDIES OF THE EFFECTS OF NITROGEN COMPOUNDS ON ANIMALS

14.1 INTRODUCTION

The toxicity of the oxides of nitrogen has been studied in a number of species including man. Previous reviews of the literature have appeared in criteria and related documents. Government-sponsored reviews include the 1971 criteria document on oxides of nitrogen (U.S. Environmental Protection Agency, 1971), the National Academy of Sciences monograph (National Academy of Sciences, 1977), the North Atlantic Treaty Organization document (North Atlantic Treaty Organization, 1973) and the USEPA document concerning short-term effects of NO_2 (U.S. Environmental Protection Agency, 1978). A World Health Organization monograph has been recently published (World Health Organization, 1977), as have two excellent interpretive reviews by Coffin and Stokinger (1977) and by Morrow (1975). The reader is referred to these publications for a general background on the toxicity of the oxides of nitrogen (NO_x).

Most of the data presented in this chapter relate to nitrogen dioxide (NO_2) because it appears to be the most toxic oxide of nitrogen and most widely distributed in a manner affecting human health. The data presented are confined to animal studies as they may relate to human health.

The focus of this chapter is to present information relating to effects on animals as a result of exposures to nitrogen oxides and other nitrogen-containing compounds at concentrations below 9,400 $\mu g/m^3$ (5 ppm). Data derived from exposures to higher concentrations of these compounds have been presented in concise form. Except for a few instances, these data are presented in the tables only.

14.2 NITROGEN DIOXIDE

14.2.1 Respiratory Tract Transport and Absorption

Nitrogen dioxide is soluble and can be absorbed in the mucous lining of the nasopharyngeal cavity where it converts to nitrous and nitric acid. However, few data examining respiratory tract uptake and transformation have been published. (See Table 14-1.) Yokoyama (1968) used isolated upper airways of the dog and rabbit to measure NO_2 removal, which amounted to 42.1 percent of the incoming NO_2 concentration. Dalhamn and Sjöholm (1963) measured the concentration of NO_2 in a stream of water-saturated air before and after it had been passed through the nose and out a tracheal cannula inserted in an anesthetized rabbit. Considerable variation occurred between animals, but about 50 percent of the incoming NO_2 was removed on a single passage through the nasopharyngeal cavity.

Goldstein et al. (1977b) exposed monkeys for 9 minutes to 560 to 1,710 $\mu g/m^3$ (0.3 to 0.91 ppm) NO_2 plus $^{13}NO_2$. During quiet respiration, 50 to 60 percent of the inspired pollutant was retained by the animal; radioactivity was distributed throughout the lungs. Once absorbed, NO_2 or chemical intermediates derived from NO_2 remained within the lung for prolonged periods following exposure. ^{13}N-radioactivity was detectable in extrapulmonary sites as well. The authors postulated that NO_2 reacted with water in the nasopharynx and lungs to form nitric and nitrous acids which then react with pulmonary and extrapulmonary tissues.

TABLE 14-1. RESPIRATORY TRACT TRANSPORT AND ABSORPTION

NO_2 Concentration μg/m³	NO_2 Concentration ppm	Duration (min)	Species	Effect	Reference
Not Reported	Not Reported	Not Reported	Dog and rabbit	Removal of 42.1% by isolated upper airways. Concentration and flow rates not given.	Yokoyama, 1968
560 to 1,710	0.3 to 0.91	9	Monkey	Concurrent exposure to $^{13}NO_2$ demonstrated that NO_2 was evenly distributed in the lungs and absorbed into the blood.	Goldstein et al., 1977b
188,000	100	≤ 45	Rabbit	Absorption of approximately 50% NO_2 in the nasopharyngeal cavity.	Dalhamn and Sjohölm, 1963

Observed effects of exposure to much higher concentrations of NO_2 include cessation of cilia beating (Dalhamn and Sjöholm, 1963) and detection of nitrates and nitrites in urine of animals (Svorcova and Kaut, 1971).

14.2.2 Mortality

In a survey of the acute toxicity of NO_2 to mice, rats, guinea pigs, rabbits, and dogs, Hine et al. (1970) found that concentrations below 94,000 $\mu g/m^3$ (50 ppm) rarely produced mortality at exposures up to 8 hours. The effects varied from species to species. (See Table 14-2.) Dogs and rabbits appear to be relatively resistant to acute toxicity. In rats, factors associated with increased mortality at high concentrations of NO_2 include cold stress and adrenalectomy.

Dietary supplementation of Vitamin E (45 to 100 mg d,l-α-tocopherol) has been shown to protect against mortality and increase mean survival of animals exposed, for long periods of time, to high concentrations of NO_2 (37,600 to 62,000 $\mu g/m^3$; 20 to 33 ppm) (Fletcher and Tappel, 1973; Menzel et al., 1972). The influence of dietary components on NO_2 toxicity is discussed more fully in Section 14.2.3.2.1.

14.2.3 Pulmonary Effects

14.2.3.1 Host Defense Mechanisms--In the past, environmental toxicologists have been concerned with measurement and description of effects of single toxic agents such as SO_2 (Fairchild et al., 1972), NO_2 (Coffin et al., 1976; Ehrlich, 1963; Purvis and Ehrlich, 1963), and O_3 (Coffin and Gardner, 1972) on host defense mechanisms. Accumulation of considerable information indicates a pathophysiological interrelationship between exposure to atmospheric pollutants and enhanced respiratory susceptibility to bacterial and viral infections.

Due to practical difficulties associated with the study of the effects of air pollutants on the susceptibility of the human respiratory system to microbial infection, animal exposures have been undertaken and several models used.

Normally, the lungs are protected from viral and bacterial infection by the combined activities of the mucociliary, phagocytic (alveolar macrophage), and immune systems. The mucociliary system, which extends from the nares to the terminal bronchioles, removes 50 to 90 percent of deposited particles within hours after they enter the lung (Cooper et al., 1977; Murphy, 1964). It is important to emphasize that the discontinuous nature of the mucous blanket precludes complete cleansing of microbes and particles from the bronchial tree. Surviving microbes and residual particles are phagocytized, killed, and/or removed by macrophages that are attracted to the foreign bodies by chemotactic factors. Microorganisms, upon entrance to the lung, also stimulate the formation of various humoral defense mechanisms. Interference, by NO_2, with any of the interdependent steps in this complex sequence of humoral and phagocytic reactions or with components of the mucociliary transport system increases host susceptibility to infection.

14.2.3.1.1 Interaction with infectious agents. Extensive studies using the infectivity model to examine the effect of NO_2 on susceptibility to airborne infection are reviewed by Ehrlich

TABLE 14-2. MORTALITY FROM NO_2 EXPOSURE FOR 1 TO 8 HOURS

NO_2 Concentration µg/m³	NO_2 Concentration ppm	Duration (hr)	Species	Effect	Reference
94,000 to 141,000	50 to 75	4 or 8	Rat	Increased mortality with cold stress, adrenalectomy, and exercise. No increase with heat or prior NO_2	Hine et al., 1970
141,000	75	1 to 8	Rat	Estimated LT50* 3.7 hr	Hine et al., 1970
141,000	75	1 to 8	Guinea pig	Estimated LT50 4.0 hr	Hine et al., 1970
141,000	75	1 to 8	Rabbit	Estimated LT50 2.7 hr	Hine et al., 1970
141,000	75	1 to 8	Dog	Estimated LT50 >8 hr	Hine et al., 1970
141,000	75	1 to 8	Mouse	Estimated LT50 2.3 hr	Hine et al., 1970
173,000	92	≤ 8	Mouse	Genetic effects on mortality of inbred mouse strains. LT50 for CF1, 3.33 hr; C57BL/6, 6.53 hr	Goldstein et al., 1973a

*LT50 = Time at which 50% of the animals would die during continuous exposure to the indicated concentration.

(1975), Coffin et al. (1976) and Gardner and Graham (1976). (See Table 14-3.) The infectivity model system has been employed successfully with hamsters (Ehrlich, 1966), mice (Coffin and Gardner, 1972; Coffin et al., 1976; Ehrlich, 1963; Fairchild et al., 1972; Purvis and Ehrlich, 1963), and squirrel monkeys (Henry et al., 1970). Animals are randomly selected for exposure to either a test substance in air (in this case NO_2) or filtered air. After exposure, control and exposed animals are placed in another chamber and exposed for a brief period (approximately 15 minutes) to aerosols of a specific infectious agent, such as *Streptoccoccus pyogenes* (*S. pyogenes*), *Klebsiella pneumoniae* (*K. pneumoniae*), *Diploccoccus pneumoniae* (*D. pneumoniae*), influenza A_2/Taiwan virus, or A/PR/8 influenza virus. The animals are then returned to clean air for a 15-day holding period and the mortality rates in the NO_2-exposed and control groups are compared. The mortality of the control group is usually 15 to 20 percent. Death is due to pneumonia and its consequences (Gardner and Graham, 1976).

In a series of investigations, the relationships of concentration and time to susceptibility to respiratory infection and to subsequent mortality in infections with *S. pyogenes* were examined (Gardner et al., 1977b; Gardner et al., 1977a, Coffin et al., 1977). The concentrations of NO_2 were varied from 1,880 to 26,320 $\mu g/m^3$ (1 to 14 ppm), and the duration of exposure ranged from 0.5 to 7 hours so that the product of concentration and time equalled a value of 7. Exposure to high concentrations of NO_2 for brief periods of time resulted in more severe infections and in greater mortality than did prolonged exposures to lower concentrations of NO_2. This indicated that susceptibility to infection was influenced more by concentration of NO_2 than by duration of exposure. (See Table 14-4.)

As depicted in Figure 14-1, Gardner et al. (Gardner et al., 1977b), using the same model, examined the effect of varying durations of continuous exposure on the mortality of mice exposed to 6 constant concentrations of NO_2 (940 $\mu g/m^3$ to 52,670 $\mu g/m^3$; 0.5 to 28 ppm). *S. pyogenes* was used for all concentrations, except 940 $\mu g/m^3$ (0.5 ppm), in which case *K. pneumoniae* was used. A linear dose-response ($p < 0.05$) indicated that mortality increases with increasing length of exposure to a given concentration of NO_2. Mortality also increased with increasing concentration of NO_2. When C x T was held constant, the relationship between concentration and time produced significantly different mortality responses. At a constant C x T of approximately 21 (ppm x hour), a 14-hour exposure at 2,800 $\mu g/m^3$ (1.5 ppm) NO_2 increased mortality by 12.5 percent whereas a 1.5-hour exposure at 27,300 $\mu g/m^3$ (14 ppm) NO_2 enhanced mortality by 58.5 percent. These studies confirmed the previous conclusion that concentration is more important than time in determining the degree of injury induced by NO_2 in this model. According to Larsen et al. (1979), NO_2 modeling studies have shown that the concentration (c) of NO_2 expected to cause a certain mortality level (z) as a function of the hours of exposure (t) can be expressed as $c = 9.55 (2.42)^z t^{-0.33}$.

Gardner et al. (1979) also compared the effect of continuous versus intermittent exposure to NO_2 followed by bacterial challenge with *S. pyogenes* (Figures 14-2, 14-3). Mice were exposed either continuously or intermittently (7 hours/day, 7 days/week) to 2,800 $\mu g/m^3$ or 6,600 $\mu g/m^3$ (1.5 or 3.5 ppm) NO_2. Figure 14-2 illustrates the results of continuous and

TABLE 14-3. INTERACTION WITH INFECTIOUS AGENTS

NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Infective Agent	Effect	Reference
560 to 940	0.3 to 0.5	Continuous, 3 mo	Mouse	A/PR/8 virus	High incidence of adenomatous proliferation of peripheral and bronchial epithelial cells. NO_2 alone & virus alone caused less severe alterations.	Motomiya et al., 1973
		Continued 3 mo more			No enhancement of effect of NO_2 and virus.	
940	0.5	Intermittent 6 or 18 hr/day, to 12 mo	Mouse	*K. pneumoniae*	Increased mortality after 6 mo intermittent exposure or after 3, 6, 9, or 12 mo continuous exposure. Following 12 mo exposure, increased mortality was significant only in continuously exposed mice.	Ehrlich and Henry, 1968
		Continuous, 90 days				
940 to 1,880	0.5 to 1	Continuous 39 days	Mouse, female	A/PR/8 virus	Significantly increased susceptibility to infection.	Ito, 1971
18,800	10	2 hr/day, 1, 3, 5 days				
1,880	1	17 hr	Mouse	*S. aureus* after NO_2	Bactericidal activity unchanged.	Goldstein et al., 1973b
4,320	2.3				6% decrease in bactericidal activity ($p<0.05$).	
12,400	6.6				35% decrease in bactericidal activity ($p<0.01$).	
2820 to 52,670	1.5 to 28	(See Figure 14-1)	Mouse	*S. pyogenes*	Increased mortality with increased time and concentration.	Gardner et al., 1979

TABLE 14-3. (continued)

NO_2 Concentration μg/m^3	 ppm	Exposure	Species	Infective Agent	Effect	Reference
1,880 5,600	1 3	3 hr	Mouse	*S. pyogenes*	Exercise on continuously moving wheels during exposure increased mortality at 5,600 μg/m^3 (3 ppm) NO_2.	Illing et al., 1980
2,820	1.5	Continuous or intermittent 7 hr/day, 7 days/wk	Mouse	*S. pyogenes*	After 1 wk, mortality with continuous exposure greater ($p < 0.05$) than that for intermittent. After 2 wk, no significant difference between continuous and intermittent exposure.	Gardner et al., 1979
6,600	3.5	Continuous or intermittent 7 hr/day, 7 days/wk, to 15 days			Increased mortality with increased duration of exposure. No significant difference between continuous and intermittent exposure. With data adjusted for total difference in C X T, mortality essentially the same.	
8,100	4.5	1, 3.5 or 7 hrs	Mouse	*S. pyogenes*	Mortality proportional to duration when bacterial challenge was immediately, but not 18 hrs post exposure	Gardner et al., (1981)
2,800 (8,100 spike	1.5 4.5)	Cont. 62 hrs. then spike for 1, 3.5 or 7 hr., then cont. 18 hrs.	Mouse	*S. pyogenes*	Mortality increased with 3.5 and 7 hr. single spike when bacterial challenge was immediately or 18 hrs post exposure	Gardner et al., (1981)

TABLE 14-3. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Infective Agent	Effect	Reference
2,800 (8,100	1.5 4.5)	Cont. for 14 d spike 2 x 1 hr/d 5 days/wk x 2 wk	Mouse	S. pyogenes	1 wk or 2 wk needed for increased mortality depending on time of infection (See text for details)	Gardner et al., (1981)
continuous:						
100 100	0.05+ 0.05 O_3	Cont.	Mouse	S. pyogenes	No effect	Gardner et al., (1981)
with spikes 2x per day:						
200 200 O_3	0.1+ 0.1 O_3	15 days (spikes-1 hr, twice/day, 5 days/wk)				
continuous:						
940 100 O_3	0.5 0.05 O_3		Mouse	S. pyogenes	Increased mortality with NO_2 alone; no effect O_3 alone; synergistic effect O_3 + NO_2	Gardner et al., (1981)
with spikes 2 x per day:						
1,880 200 O_3	1.0+ 0.1 O_3					
continuous:						
2,300 200	1.2+ 0.1 O_3	Cont. 15 days (spikes-1 hr, twice/day, 5 days/wk)	Mouse	S. pyogenes	Increased mortality with NO_2 alone and O_3 alone; additive effect of O_3 + NO_2	Gardner et al., (1981)
with spikes 2 x per day:						
4,700 600 O_3	2.5 0.3 O_3					
2,800	1.5	Cont.	Mouse	S. pyogenes	Elevated temp. (32° C) increased mortality	Gardner et al., (1981)

TABLE 14-3. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Infective Agent	Effect	Reference
3,570	1.9	4 hr	Mouse	Infected with _S_. _aureus_ prior to NO_2 exposure	Physical removal of bacteria unchanged at 3,570 and 7,140 µg/m³ (1.9 and 3.8 ppm).	Goldstein et al., 1973b
7,140	3.8					
13,160	7				7% lower bactericidal activity ($p<0.05$).	
17,200	9.2				14% lower bactericidal activity ($p<0.01$).	
27,800	14.8				50% lower bactericidal activity ($p<0.01$).	
3,760	2	3 hr	Mouse	_S_. _pyogenes_	Increased mortality ($p<0.05$)	Ehrlich et al., 1977
4,700	2.5	2 hr	Mouse	Challenge with _K_. _pneumoniae_ before and after exposure	No effect on mortality. At 47,000 µg/m³ (25 ppm) effect when bacterial challenge was up to 72 hrs. but not later, after NO_2 exposure ceased.	Purvis and Ehrlich, 1963
47,000	25					
9,400	5				Significant increase in mortality on _K_. _pneumoniae_ challenge 1 and 6 hr post NO_2. When _K_. _pneumoniae_ challenge 27 hr post NO_2 effect only at 28,200 µg/m³ (15 ppm).	
18,800	10					
28,200	15					
6,580	3.5	2 hr	Mouse	_K_. _pneumoniae_ challenge after exposure	NO_2 toxic to all species and increased mortality. Each species had decreased resistance to NO_2 and bacteria.	Ehrlich, 1975
65,830	35	2 hr	Hamster			

TABLE 14-3. (continued)

NO$_2$ Concentration µg/m^3	NO$_2$ Concentration ppm	Exposure	Species	Infective Agent	Effect	Reference
94,050	50	2 hr	Squirrel monkey			
9,400	5	Continuous, 2 mo	Squirrel monkey	*K. pneumoniae* and *A/PR/8* virus	One-third died after infection.	Henry et al., 1970
19,000	10	Continuous 1 mo			Death within 2-3 days after infection. Increased susceptibility to infection. Decreased lung clearance of viable bacteria.	
9,400	5	2 mo	Squirrel monkey	*K. pneumoniae*	Mortality 2/7. Bacteria present in lung of survivors upon autopsy.	Henry et al., 1969
19,000	10	1 mo			Mortality 1/4. Bacteria present in lungs of survivors at autopsy.	
94,000	50	2 hr			Mortality 3/3.	

TABLE 14-4. THE INFLUENCE OF CONCENTRATION AND TIME ON EHNANCEMENT OF MORTALITY RESULTING FROM VARIOUS NO_2 CONCENTRATIONS[a]

		Concentration x time					
		7		14		21	
Concentration µg/m³	ppm	Time (hrs)	% Mortality	Time (hrs)	% Mortality	Time (hrs)	% Mortality
2,820	1.5	4.7	6.4	9.3	10.2	14.00	12.5
6,580	3.5	2.0	18.7	4.0	27.0	6.00	31.9
13,160	7	1.0	30.2	2.0	41.8	3.00	48.6
26,320	14	.5	21.7	1.0	44.9	1.50	58.5
52,640	28	.25	55.5	.5	67.2	.75	74.0

[a] These are predicted values obtained from Figure 1 of Gardner et al., 1979.

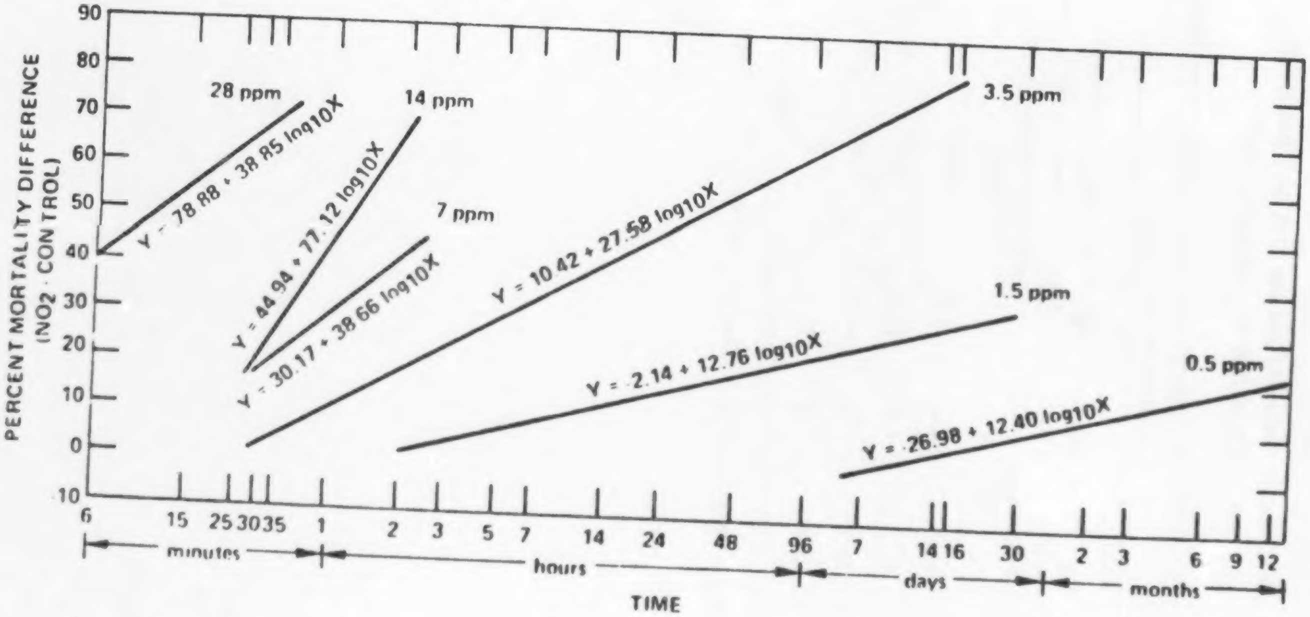


Figure 14-1. Regression lines of percent mortality of mice versus length of continuous exposure to various NO_2 concentrations prior to challenge with bacteria (Gardner et al., 1977b).

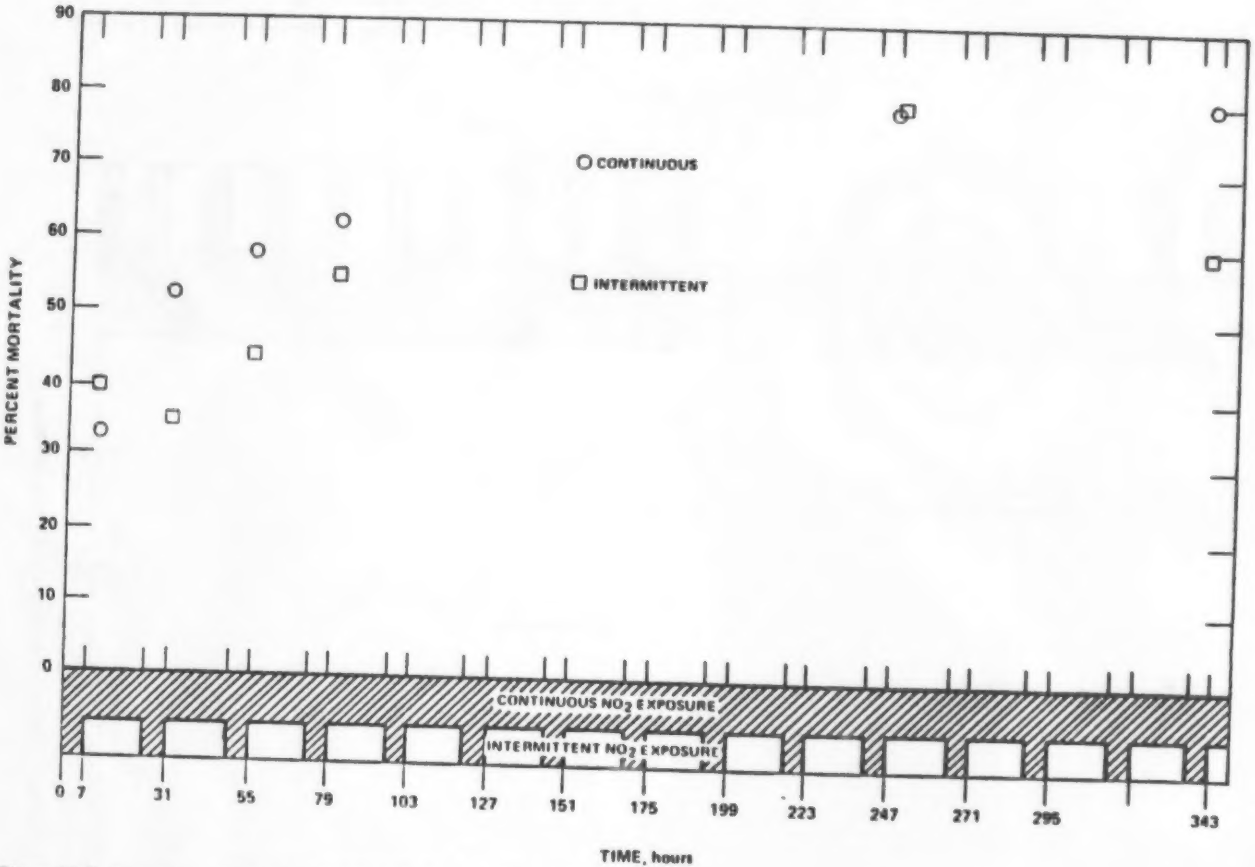


Figure 14-2. Percent mortality of mice versus the length of either continuous or intermittent exposure to 6,600 μg/m^3 (3.5 ppm) NO_2 prior to challenge with streptococci (Gardner et al., 1977b; Gardner et al., 1977a; Coffin et al., 1977).

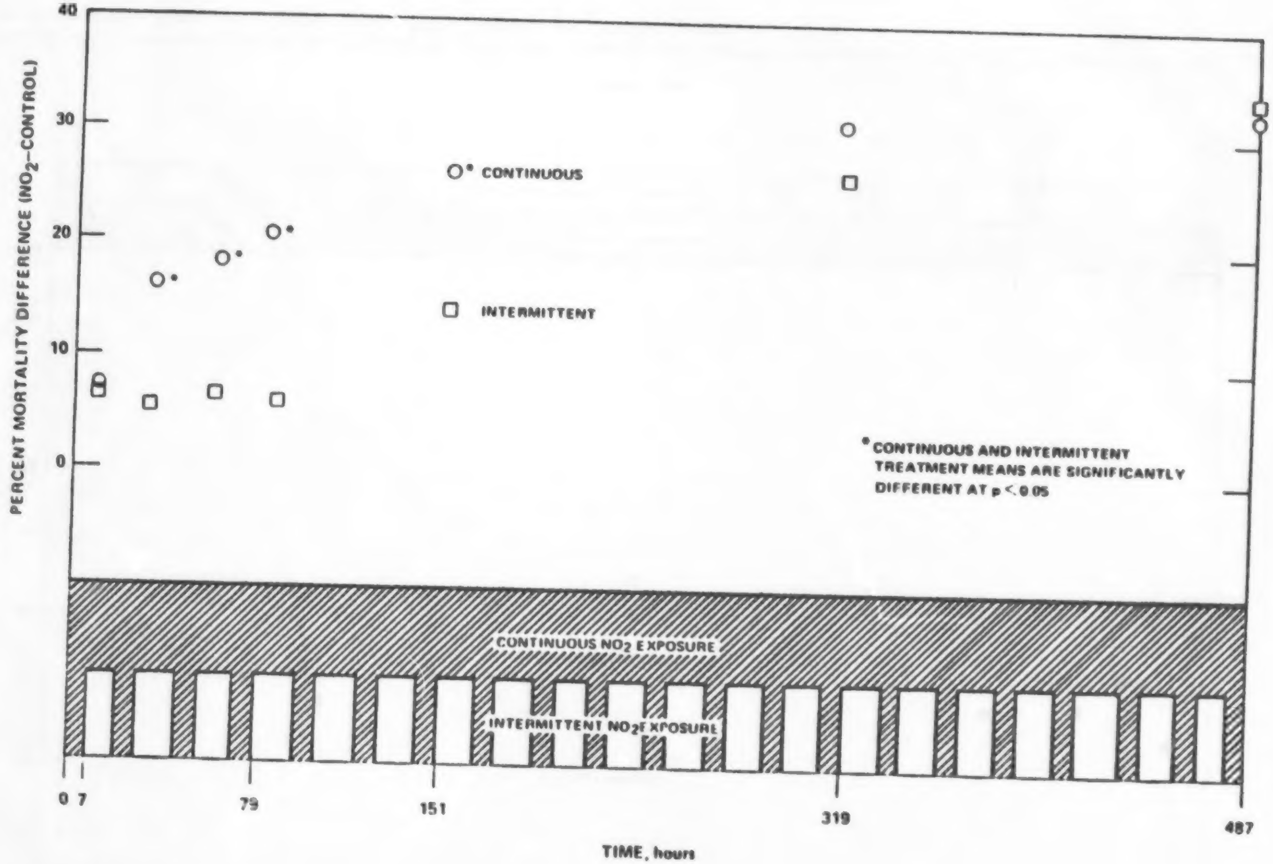


Figure 14-3. Percent mortality of mice versus length of either continuous or intermittent exposure to 2,800 $\mu g/m^3$ (1.5 ppm) NO_2 prior to challenge with streptococci (Gardner et al., 1977b; Gardner et al., 1977a; Coffin et al., 1977).

intermittent exposure to 6,600 μg/m^3 (3.5 ppm) NO_2 for periods up to 15 days. There was a significant increase in mortality for each of the experimental groups with increasing duration of exposure. When the data were adjusted for the difference in C x T, the mortality was essentially the same for the continuous and intermittent groups. The continuous exposure of mice to 2,800 μg/m^3 (1.5 ppm) NO_2 increased mortality after 24 hours of exposure. During the first week of exposure, the mortality was significantly higher in mice exposed continuously to NO_2 than in those exposed intermittently. By the 14th day of exposure, the difference between intermittent and continuous exposure became indistinguishable (Figure 14-3).

Mice were exposed continuously or intermittently (6 or 18 hours/day) to 940 μg/m^3 (0.5 ppm) NO_2 for up to 12 months (Ehrlich and Henry, 1968). Neither exposure regimen affected murine resistance to K. pneumoniae infection during the first month. Those exposed continuously exhibited decreased resistance to the infectious agent as demonstrated by enhanced ($p < 0.05$) mortality at 3, 6, 9, and 12 months. In another experiment, an enhancement ($p < 0.1$) did not occur at 3 months but was observed after 6 months of exposure. After 6 months, mice exposed intermittently (6 or 18 hours/day) to NO_2 showed significant ($p < 0.1$) increases in mortality over that of controls (18%). After 12 months exposure to NO_2, mice in the three experimental groups showed a reduced capacity to clear viable bacteria from the lung. This was first observed after 6 months in the continuously exposed mice and after 9 months in the two intermittently exposed groups. These changes, however, were not statistically tested for significance. Only the continuously exposed animals showed increased mortality (23%) over controls following 12 months exposure. Therefore, while it is not possible to directly compare the results of studies using S. pyogenes to those using K. pneumoniae, the data suggest that as the concentration of NO_2 is decreased, a longer exposure time is necessary for the intermittent exposure regimen to produce a level of effect equivalent to that of continuous exposure.

Gardner et al. (1981) investigated further the effects of intermittent exposures on the response of mice to airborne infections. The objective of these studies was to investigate the toxicity of spikes of NO_2 exposure superimposed on a lower continuous NO_2 exposure. Such a regimen approximates the pattern of exposure which man receives in urban environments. Mice were exposed to spikes of 8,100 μg/m^3 (4.5 ppm) for 1, 3.5 or 7 hrs and exposed to S. pyogenes either immediately or 18 hrs afterwards. Mortality was proportional to the duration of the spike when mice were exposed immediately, but mice recovered from the exposure by 18 hrs. When these same spikes were superimposed on a continuous background of 2,800 μg/m^3 (1.5 ppm) for 62 hrs preceding and 18 hours following the spike, mortality was significantly enhanced ($p < 0.05$) only by a spike of 3.5 or 7 hrs when the infectious agent was administered 18 hours after the spike. Possible explanations for these differences in the presence or absence of a background exposure are that mice continuously exposed are not capable of recovery or that alveolar macrophages or polymorphonuclear leukocytes recruited to the site of infection are impaired by the continuous exposure to NO_2. The effect of multiple spikes was examined by exposing mice for 2 weeks to two daily spikes (morning and afternoon) of 1 hr of 8,100 μg/m^3 (4.5 ppm) superimposed to a continuous background of 2,800 μg/m^3 (1.5 ppm). Spikes were not superimposed on the continuous background during weekends. Mice were exposed to the infectious agent either immediately before or after the morning spike. When the infectious agent was given before the morning spike, the increase in mortality did not approach closely that of a continuous exposure to 2800 μg/m^3 (1.5 ppm). However, in mice exposed after the morning spike, by 2 weeks of exposure, the increased mortality over controls approached that equivalent to continuous exposure to 2,800 μg/m^3 (1.5 ppm).

In this same study, Gardner et al. (1981) also examined the effects of exposure to spikes of O_3 and NO_2, and heat stress. At the lowest concentration of O_3 and NO_2 examined, 100 μg/m^3 (0.05 ppm) with spikes of 200 μg/m^3 (0.1 ppm), no differences in mortality were observed compared to clean air controls. When mice were exposed to intermediate (0.5 ppm NO_2 with 1.0 ppm spikes and 0.05 ppm O_3 with 0.1 ppm spikes) or high (1.2 ppm NO_2 with 2.5 ppm spikes and 0.1 ppm O_3 with 0.3 ppm spikes) doses, mortality was increased synergistically; e.g., mortality was greater than the arithmetic sum of the mortality due to the single gas exposure. When mice were stressed by elevated temperature (32°C), 7 daily, but not 4 daily, exposures to 2,800 μg/m^3 (1.5 ppm) enhanced mortality and decreased survival times.

Another stress, exercise, was also evaluated with the infectivity model (Illing et al., 1980). Mice running on an activity wheel during exposure were more susceptible to 5,600 μg/m^3 (3.0 ppm) than those resting.

Gardner et al. (1981) concluded that while a simple log-log relationship exists for the mortality associated with a given exposure-time product with mice continuously exposed to NO_2 or given intermittent regular exposures, no such relationship exists for mice continuously exposed to a constant concentration of NO_2 upon which are superimposed spikes of greater concentration. The relationship is highly complex depending in part upon the duration of the spike and the time since the last exposure of the spike.

Mice, hamsters, and monkeys were exposed to NO_2 for 2 hours followed by a challenge of _K. pneumoniae_ (Ehrlich, 1975). Nitrogen dioxide enhanced the mortality due to the pathogen in all species tested. Differing results among species were found. This could be due to differing sensitivity to either the pathogen or NO_2, or a combination of both. All three squirrel monkeys exposed to 94,050 μg/m^3 (50 ppm) NO_2 died from the pneumonia (Henry et al., 1969). Lower concentrations tested (9,400 to 65,830 μg/m^3; 5 to 35 ppm) had no effect in monkeys. The hamster model, which exhibited enhanced mortality due to NO_2 at concentrations $\geq$ 65,830 μg/m^3 (35 ppm) but not at 9,400 to 47,000 μg/m^3 (5 to 25 ppm), had intermediate sensitivity. The mouse model was sensitive to NO_2 exposure as evidenced by enhanced mortality following exposure to 6,580 μg/m^3 (3.5 ppm) but not to 2,820 to 4,700 μg/m^3 (1.5 to 2.5 ppm) NO_2 for 2 hours (Ehrlich, 1975). No effect on mortality was observed in mice exposed for 2 hours to 4,700 μg/m^3 (2.5 ppm) (Purvis and Ehrlich, 1963). However, when _S. pyogenes_ was the infectious agent, a 3-hour exposure to 3,760 μg/m^3 (2 ppm) NO_2 caused an increase ($p < 0.05$) in mortality (Ehrlich et al., 1977).

The persistence of the NO_2 effect was investigated by Purvis and Ehrlich (1963) who exposed mice for 2 hours to NO_2 before or after an aerosol challenge with K. pneumoniae. At 9,400, 18,800, 28,200 and 47,000 µg/m^3 (5, 10, 15, or 25 ppm) NO_2, there was a significant enhancement of mortality in mice challenged with bacteria 1 and 6 hours after the NO_2 exposure. When bacterial challenge was delayed for 27 hours, there was an effect only in the group exposed to 28,200 µg/m^3 (15 ppm). Exposure to 4,700 µg/m^3 (2.5 ppm) caused no effect at any of the bacterial challenge times tested. Exposure of 47,000 µg/m^3 (25 ppm) NO_2 for 2 hours with subsequent K. pneumoniae challenge 6 and 14 days later did not affect mortality. When the experimental regimen was reversed and mice were exposed for 2 hours to 47,000 µg/m^3 (25 ppm) NO_2, mortality was significantly increased 1, 6, 27, 48 and 72 hours after bacterial challenge. Dose response studies in which mice were challenged 1 hour after a 2 hour NO_2 exposure showed that 6,580 µg/m^3 (3.5 ppm) had a significant effect; exposure concentrations of 2,820 and 4,700 µg/m^3 (1.5 and 2.5 ppm) did not.

Environmental stress has been shown to enhance the toxic effect of NO_2. Mice placed on continuously moving exercise wheels during exposure to 5,600 µg/m^3 (3 ppm) NO_2, but not 1,880 µg/m^3 (1 ppm), for 3 hours showed enhanced mortality over nonexercised NO_2 exposed mice ($p < 0.06$) using the infectivity model (Illing et al., 1980). The presence of other environmental factors, ozone (O_3) (Ehrlich et al., 1977, Gardner et al., 1981) or tobacco smoke (Henry et al., 1971), also augments the deleterious effect of NO_2 on host resistance to experimental infection (see Section 14.3).

Squirrel monkeys exposed continuously to NO_2 levels of 18,800 µg/m^3 and 9,400 µg/m^3 (10 ppm and 5 ppm) in air for 1 and 2 months, respectively, showed increased susceptibility to a challenge with K. pneumoniae or influenza A/PR/8 virus and reduced lung clearance of viable bacteria (Henry et al., 1970). All six animals exposed to 18,800 µg/m^3 (10 ppm) died within 2 to 3 days of infection with the influenza virus. At 9,400 µg/m^3 (5 ppm), one of three monkeys died. Susceptibility to viral infection also was enhanced when the NO_2 exposure occurred 24 hours after infectious challenge. Exposure to 94,000 µg/m^3 (50 ppm) NO_2 for 2 hours was not fatal, whereas the same exposure followed by challenge with K. pneumoniae was fatal to three out of three monkeys (Henry et al., 1969). After challenge with K. pneumoniae, two of seven monkeys exposed to 9,400 µg/m^3 (5 ppm) for 2 months died and the rest had bacteria in the lungs on autopsy. After an exposure to 18,800 µg/m^3 (10 ppm) for 1 month, one of four monkeys died, and the pathogen was present in the lungs of the remainder of the animals at autopsy 19 to 51 days post-exposure.

Mice exposed continuously for 3 months to 560 to 940 µg/m^3 (0.3 to 0.5 ppm) NO_2 followed by challenge with A/PR/8 influenza virus demonstrate significant pulmonary pathological responses. Motomiya et al. (1973) reported a greater incidence of adenomatous proliferation of bronchial epithelial cells following combined exposures; viral exposures or NO_2 alone caused less severe alterations than the combination of NO_2 plus virus. Continuous NO_2 exposure for an additional 3 months did not enhance further the effect of NO_2 or the subsequent virus challenge.

Ito (1971) challenged mice with influenza A/PR/8 virus after continuous exposure to 940 to 1,880 μg/m^3 (0.5 to 1 ppm) NO_2 for 39 days and to 18,800 μg/m^3 (10 ppm) NO_2 for 2 hours daily for 1, 3, and 5 days. Acute and intermittent exposure to 18,800 μg/m^3 (10 ppm) NO_2 as well as continuous exposure to 940 to 1,880 μg/m^3 (0.5 to 1 ppm) NO_2 increased the susceptibility of mice to influenza virus as demonstrated by increased mortality.

The enhancement in mortality following exposure to NO_2 and a pathogenic organism could be due to several factors. One could be a decreased ability of the lung to kill bacteria. Studies by Goldstein et al. (1973b, 1974) illustrated this concept in a series of experiments which show decreased bactericidal activity following exposure to various pollutants. In the first experiments, mice breathed aerosols of *Staphylococcus aureus* (*S. aureus*) labelled with radioactive phosphorus (^{32}P) and were then exposed to NO_2 for 4 hours (Goldstein et al., 1973b). Physical removal of the bacteria was not affected by any of the NO_2 concentrations used up to 27,800 μg/m^3 (14.8 ppm). Concentrations of 13,200, 17,200, and 27,800 μg/m^3 (7, 9.2, and 14.8 ppm) NO_2 lowered bactericidal activity by 7, 14, and 50 percent, respectively, when compared to controls ($p < 0.05$). Lower concentrations (3,570 and 7,140 μg/m^3; 1.9 and 3.8 ppm) had no significant effect. In another experiment, mice breathed NO_2 for 17 hours and then were exposed to an aerosol of *S. aureus*. Four hours later the animals were examined for the amount of bacteria present in their lungs. No difference in the inhalation of bacteria was found with NO_2 exposure. Concentrations of 4,320 and 12,400 μg/m^3 (2.3 and 6.6 ppm) NO_2 decreased pulmonary bactericidal activity by 6 and 35 percent, respectively, compared to control values ($p < 0.05$). Exposure to 1,880 μg/m^3 (1 ppm) NO_2 had no significant effect. Goldstein et al. hypothesized that the decreased bactericidal activity was due to defects in alveolar macrophage function. The detailed effects of NO_2 exposure on the function of alveolar macrophages are presented in Section 14.2.3.1.3.

14.2.3.1.2 *Mucociliary transport*. Mucociliary transport is the principal mechanism for removal of inspired and aspirated particles from the tracheobronchial tree. Concentrations of NO_2 greater than 9,400 μg/m^3 (5 ppm) decrease rates of ciliary beating as measured *in vitro* (Kita and Omichi, 1974) and of mucociliary transport *in vivo* (Giordano and Morrow, 1972). The effect of lower concentrations of NO_2 on mucociliary function is unknown. (See Table 14-5)

Schiff (1977) exposed hamster tracheal ring cultures to 3,760 μg/m^3 (2 ppm) NO_2 for 1.5 hours/day, 5 days/week, for 1, 2, and 3 weeks. Tracheal cultures infected with influenza virus immediately after the initiation of the NO_2 exposure were not different from control infected cultures. However, explants infected after 1 or 2 weeks of NO_2 exposure showed decreased ciliary activity and morphological changes compared to controls held in filtered air. After 14 days exposure to NO_2 non-infected cultures showed a decrease in ciliary activity and morphological changes. After 4 weeks exposure of the uninfected cultures, there was a 63 percent decrease in ciliary activity. In addition, tracheal organ cultures exposed to NO_2 exhibited a more rapid production of virus than explants held in filtered air.

14.2.3.1.3 *Alveolar macrophage*. Exposures of animals to NO_2 concentrations ranging from 13,160 to 112,800 μg/m^3 (7 to 60 ppm) cause a variety of structural and physiological

TABLE 14-5. MUCOCILIARY TRANSPORT

NO_2 Concentration µg/m^3	ppm	Exposure	Species	Effect	Reference
11,280	6	7 days/wk, 6 wk	Rat, female	Increase in TPTT and FET[a]: decrease in mucociliary velocity, $p<0.02$. Functional impairment reversed within 1 wk.	Giordano and Morrow, 1972

[a] TPTT = Twenty percent transport time.
FET = First-edge time.

abnormalities in alveolar macrophages. (See Table 14-6.) Alveolar macrophages (AM) isolated from animals exposed to these concentrations of NO_2 show diminished phagocytic activity, (Gardner et al., 1969) appearance of intracellular dense bodies, (Katz and Laskin, 1975) increased congregation of AM on epithelial cells, (Sherwin et al., 1968) enhanced wheat germ lipase-induced binding of autologous and heterologous red blood cells to AM, (Hadley et al., 1977) increased _in vitro_ penetration of AM by virus, (Williams et al., 1972) reduced _in vitro_ production of interferon, (Valand et al., 1970) and increased mitochondrial and decreased cytoplasmic NAD+/NADH ratios. (Mintz, 1972; Simons et al., 1974) An increased number of polymorphonuclear leukocytes was observed in lung lavages of animals exposed to high levels of NO_2 (Gardner et al., 1969). (Table 14-6.)

Aranyi et al. (1976) used scanning electron microscopy (SEM) to study the effect of exposure to NO_2 on the anatomic integrity of mouse alveolar macrophages which were lavaged from the lung. No changes in the AM surface were observed after continuous exposure of mice for 4, 12, and 24 weeks to 940 $\mu g/m^3$ (0.5 ppm) without peaks or 188 $\mu g/m^3$ (0.1 ppm) NO_2 with 3-hour peaks at 1,880 $\mu g/m^3$ (1 ppm) for 5 days/week. Macrophages from mice continuously exposed to 3,760 $\mu g/m^3$ (2 ppm) without peaks or 940 $\mu g/m^3$ (0.5 ppm) NO_2 with 1-hour peaks of 3,760 $\mu g/m^3$ (2 ppm) NO_2 for 5 days/week, showed distinctive morphological alterations after 21 weeks total exposure. Loss of surface processes, appearance of fenestrae, bleb formation, denuded surface areas, as well as, occasionally, complete deterioration of the cells were seen. Structural changes were still observed at the same NO_2 concentrations after continuous exposure to a baseline of 940 $\mu g/m^3$ (0.5 ppm) with peaks of 3,760 $\mu g/m^3$ (2 ppm) NO_2 for 28 or 33 weeks. These observations appear to correlate well with a reduction in _in vitro_ phagocytic activity and increased susceptibility to infection.

Acton and Myrvik (1972) administered an intratracheal injection of parainfluenza-3 virus to rabbits prior to 3-hour exposures to 9,400, 28,200, 47,000, or 94,000 $\mu g/m^3$ (5, 15, 25, or 50 ppm) NO_2. Alveolar macrophages harvested from exposed, as well as control animals, were challenged with rabbitpox virus. Macrophages from control animals infected with influenza had increased resistance (75 percent) to pox virus. However, there was partial loss of resistance 48 hours following exposures to 28,200 $\mu g/m^3$ (15 ppm) NO_2. No decrease in resistance was observed with 9,400 $\mu g/m^3$ (5 ppm) NO_2. Phagocytic capabilities were adversely affected in macrophages from animals exposed to 28,200 to 94,000 $\mu g/m^3$ (15 to 50 ppm) NO_2. At a concentration of 94,000 $\mu g/m^3$ (50 ppm), NO_2 stimulated oxygen uptake and hexose monophosphate shunt activity in the AM.

Nitrogen dioxide-induced alteration of receptor sites of the alveolar macrophages has been studied by Goldstein et al. (Goldstein et al., 1977a; Goldstein, 1979). It was found that _in vitro_ exposure of rat alveolar macrophages to 4,512 $\mu g/m^3$ (2.4 ppm) NO_2 for 1 hour resulted in a 64 percent increase in agglutination by concanavalin A (Goldstein et al., 1977a). Alveolar macrophages collected from rats exposed to 22,748 $\mu g/m^3$ (12.1 ppm) NO_2 for 2 hours displayed a 40 percent increased agglutinability to concanavalin A. Following exposure of alveolar macrophages to 6,768 $\mu g/m^3$ (3.6 ppm) NO_2 for 1 hour, incubation of macrophages

TABLE 14-6. ALVEOLAR MACROPHAGES

NO_2 Concentration µg/m^3	ppm	Exposure	Species	Effect	Reference
940	0.5	Continuous exposure for 4, 12, 24 wks	Mouse	Alveolar macrophage (AM) surface unchanged.	Aranyi et al., 1976
188 [with 3 hr peaks of 1,880 µg/m^3 (1 ppm)]	0.1	Exposure for 4, 12, 24 wks, 5 days/wk			
3,760	2	Continuous exposure for 21, 28, 33 wks	Mouse	Distinct morphological alterations after 21 wk total exposure.	Aranyi et al., 1976
940 [with 1 hr peaks of 3,760 µg/m^3 (2 ppm)]	0.5	Continuous exposure for 21, 28, 33 wks, 5 days/wk		Loss of surface processes, appearance of fenestrae, bleb formation, denuded surface areas, and complete deterioration of cells were noted.	
6,768	3.6	1 hr	Rat	Incubation of macrophages with ^3H-concanavalin A revealed no significant alterations in binding. At this concentration, agglutination was enhanced 47%.	Goldstein et al., 1977a
22,748	12.1	2 hr		40 percent increase in concanavalin A agglutination of macrophages.	

TABLE 14-6. (continued)

NO_2 Concentration μg/m³	ppm	Exposure	Species	Effect	Reference
9,400	5	3 hr exposure after infection with parainfluenza-3 virus. Challenge with rabbit pox virus.	Rabbit	Control AM had increased resistance (75%) to pox virus. Partial loss of resistance, following 28,200 μg/m³ (15 ppm) NO_2.	Acton and Myrvik, 1972
28,200	15				
47,000	25			Decreased phagocytic capabilities at all concentrations except 9,400 μg/m³ (5 ppm).	
94,000	50			Reduction in resistance, decreased phagocytic capabilities, stimulation of O_2 uptake plus hexose-monophospate shunt activity.	
13,200	7	24 hr	Rabbit	Increased rosette formation in AM treated with wheat germ lipase.	Hadley et al., 1977
15,000 to 112,800	8 to 60	3 hr	Rabbit	Increased number of poly-morpho-nuclear leukocytes in lavage fluid persisted for more than 72 hr.	Gardner et al., 1969
18,800	10	24 hr	Rat	Phagocytic activity was unchanged.	Katz and Laskin, 1975
47,000	25			Depressed phagocytosis was seen on 3rd day of culture. Macrophages apparently recovered by 7th day of culture.	
18,800	10	7 wk continuous	Guinea pig	63% increase in epithelial cells positive for macrophage congregation. Presence of 7 or more AM on a single epithelial cell-2.5 times more frequent.	Sherwin et al., 1968

TABLE 14-6. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
19,000	10	3 hr	Rabbit	50% inhibition of phagocytic activity.	Gardner et al., 1969
43,300	23	1 hr	Rabbit	Increased mitochondrial and decreased cytoplasmic $NAD^+/NADH$ were observed.	Mintz, 1972
47,000	25	3 hr	Rabbit	No development of resistance with NO_2 exposure immediately after infection with parainfluenza-3 virus or up to 24 hr before viral inoculation. Increased lung absorption of virus. No effect on viral potency.	Valand et al., 1970
47,000	25	3 hr	Rabbit	Viral uptake not affected when infected with parainfluenza-3 virus after NO_2 exposure. No inhibition of viral RNA synthesis. Twice as many virus attached and penetrated exposed AM.	Williams et al., 1972

with ^{3}H-concanavalin A revealed no significant alterations in binding of ^{3}H-concanavalin A to the macrophages. At this concentration of NO_2, agglutination was enhanced 47 percent.

Green and Schneider (1978) exposed baboons to 3,760 μg/m^3 (2 ppm) NO_2 for 8 hr/day, 5 days/wk for 6 months and examined the response of their alveolar macrophages to migration inhibition factor (MIF). MIF is a substance produced by lymphocytes which inhibits migration of macrophages and thus influences their protective functions. Two of three of the antigen-sensitized, NO_2-exposed animals did not respond to MIF. Macrophages from 3 of the 4 NO_2-exposed baboons had diminished responsiveness to MIF.

Voisin et al. (1976; 1977) exposed guinea pig macrophages, in vitro, to 188, 1,880, 3,760, and 9,400 μg/m^3 (0.1, 1, 2, and 5 ppm) NO_2 for 30 minutes. The surviving cells showed decreased bactericidal activity, especially at the 9,400 μg/m^3 (5 ppm) level, as well as reduction in ATP content and changes in morphology. Following exposure to 188 μg/m^3 (0.1 ppm) NO_2, the alveolar macrophage membranes appeared to be spread out and to emit cytoplasmic projections. These projections were much more evident upon exposure to 1,880 and 3,760 μg/m^3 (1 and 2 ppm) NO_2. At 3,760 μg/m^3 (2 ppm) NO_2, the nucleus became hard to identify due to its washed-out appearance.

Vassallo et al. (1973) found that in vitro exposure to NO_2 for 15 to 20 minutes could damage the AM. Phagocytosis and bactericidal capability were adversely affected by NO_2 concentrations at 15 mM (690 ppm) ($p < 0.05$). Both 5 and 10 mM (230 and 460 ppm) NO_2 increased $^{14}CO_2$ production from ^{14}C-1- and ^{14}C-6-glucose and from ^{14}C-1-pyruvate ($p < 0.05$) in the resting AM, with similar results occurring in phagocytizing cells, except for the ^{14}C-6-glucose substrate. Nitrogen dioxide diminished the conversion of formate to CO_2 by approximately 50 percent. A concentration of 0.5 mM (23 ppm) also prevented the inhibition of AM catalase activity caused by a subsequent addition of aminotriazale, whereas NO_2 alone did not inhibit its activity.

14.2.3.1.4 Immune system. The effects of exposures of animals to NO_2 on a few parameters of the immune response have been investigated by a small number of workers (Antweiller et al., 1975; Balchum et al., 1965; Ehrlick and Fenters, 1973; Ehrlich et al., 1975; Fenters et al., 1971; Fenters et al., 1973; Kosmider et al., 1973b; Matsumura, 1970a). (See Table 14-7.) It should be emphasized that local responses within the lung are critical in regard to antimicrobial defense and that these responses are, for the most part, unstudied. Ehrlich et al. (1975) exposed male SPF Swiss albino mice continuously to 3,760 μg/m^3 (2 ppm) or 940 μg/m^3 (0.5 ppm) NO_2 with daily 1-hour peaks of 3,760 μg/m^3 (2 ppm) for 5 days/ week for 3 months. After exposure, all mice were vaccinated with influenza A_2/Taiwan/1/64. Mean serum neutralizing antibody titer was four-fold lower with NO_2 exposure ($p < 0.05$) than with controls. Control mice breathing filtered air also showed a depressed serum neutralizing antibody titer 2 weeks after vaccination ($p < 0.05$). However, 4 to 8 weeks after vaccination there were no differences between controls and treated. The hemagglutination inhibition titer was not affected. Non-vaccinated mice exposed to either NO_2 regimen had decreased serum IgA and increased serum IgG_1 ($p < 0.05$). Mice breathing 940 μg/m^3 (0.5 ppm) NO_2 with peaks of 3,760

TABLE 14-7. IMMUNOLOGICAL EFFECTS

Pollutant	Pollutant Concentration µg/m³	Pollutant Concentration ppm	Exposure	Species	Effects	Reference
NO_2	3,760	2	24 hr/day, 5 days/wk, 3 mo followed by vaccination with influenza A_2/Taiwan/1/64 virus	Mouse, male	4-fold decrease in serum neutralizing antibody titer. Hemagglutination inhibition titers unchanged. Before viral challenge, decreased serum IgA and increased serum IgG_1. Increased IgM and IgG_2 ($p < 0.05$). Serum IgA unchanged, IgM increased ($p < 0.05$) after virus.	Ehrlich et al., 1975
	940 with daily 1 hr 3,760	0.5 with daily 1 hr 2				
NO_2	1,880	1	Continuous, 493 days; challenge 5 times with monkey adapted influenza A/PR/8/34 virus during exposure	Monkey	Hemagglutination inhibition titers unchanged. Increased mean serum neutralizing antibody titers after 493 days exposure. Titers increased 7-fold 21 days post virus. Titers increased 11-fold 41 days post virus.	Fenters et al., 1973
NO_2	1,880	1	6 mo followed by intranasal challenge with D. pneumoniae	Guinea pig	Increased respiratory infection. Decreased hemolytic activity of complement. Decreased all immunoelectrophoretic fractions. Increased mortality following D. pneumoniae.	Kosmider et al., 1973b
NO_2	9,400	5	Continuous, to 169 days; challenge with mouse adapted influenza A/PR/8/34 virus 24 hr prior to exposure	Monkey	Hemagglutination inhibition titers or amnestic response unchanged. Initial depression in serum neutralization titers with return to normal by 133 days.	Fenters et al., 1971
NO_2	9,400	5	4 hr/day, 5 days/wk	Guinea pig	Lung tissue serum antibodies increased with intensity and duration of exposure.	Balchum et al., 1965
	9,400 28,200	5 15	7.5 hr/day, 5 days/wk			
NO_2	10,000	5.3	Continuous, 33 days	Guinea pig	No effect on antibody production.	Antweiller, 1975
NO_2	37,600 75,200 131,700	20 40 70	Gas 30 min, then aerosol of egg albumin or bovine serum albumin for 45 min, repeated 5-7 times on different days	Guinea pig	Anaphylactic attacks in 50% exposed animals by 5th aerosol administration at highest concentration. No effect at lower levels. Hemagglutination tests unchanged. Less antigen needed in active cutaneous anaphylaxis test at highest levels ($p < 0.05$).	Matsumura, 1970a

TABLE 14-7. (continued)

Pollutant	Pollutant Concentration		Exposure	Species	Effects	Reference
	$\mu g/m^3$	ppm				
NO_2	56,400 75,200 84,600 94,000	30 40 45 50	Exposure 30 min then nebulized acetylcholine.	Guinea pig	Mortality increased at NO_2 > 94,000 $\mu g/m^3$ (50 ppm).	Matsumura et al., 1972
NO_2	75,000 150,000	40 80	Sensitized to egg and bovine serum albumin by intraperitoneal injection; 3 days later exposure for 30 min to pollutants then antigen 30 min later.	Guinea pig	Mortality: 20% 37%	Matsumura, 1970b

μg/m^3 (2 ppm) also had increases in IgM and IgG_2 ($p < 0.05$). A different response was seen after the mice received the virus. Serum IgA increased ($p < 0.05$) only when mice were held in filtered air, vaccinated and exposed to 940 or 3,760 μg/m^3 (0.5 or 2 ppm) NO_2. Immunoglobulin M (IgM) concentrations were elevated in all NO_2-exposed groups. A significant increase ($p < 0.05$) took place in only the following groups: (a) continuous exposure to 940 or 3,760 μg/m^3 (0.5 or 2 ppm), (b) continuous exposure to 940 or 3,760 μg/m^3 (0.5 or 2 ppm) or to 3,760 μg/m^3 (2 ppm), pre-vaccination and clean air afterwards, and (c) 3 months filtered air and 3,760 μg/m^3 (2 ppm) NO_2 post-vaccination. Similar results were observed for IgG_2 and IgG_1 determinations.

The immune system of monkeys exposed to NO_2 was studied in an additional series of experiments (Ehrlich and Fenters, 1973; Fenters et al., 1971; Fenters et al., 1973). Fenters et al. (1971) injected mouse-adapted influenza A/PR/8/34 intratracheally 24 hours prior to continuous exposure to 9,400 μg/m^3 (5 ppm) NO_2. Hemagglutination inhibition titers to influenza titers were not changed. Initially serum neutralization titers were depressed by NO_2. By 133 days, the effect had disappeared. The amnestic response was not affected.

Fenters et al. (1973) described the effects of continuous exposures of 1,880 μg/m^3 (1 ppm) NO_2 for 493 days on monkeys challenged five times via intratracheal injection to live monkey-adapted influenza virus A/PR/8/34 during NO_2 exposure. Again, hemagglutination inhibition titers were not significantly affected by NO_2 exposures. However, the mean serum neutralizing antibody titers were significantly higher in animals exposed to NO_2 for 493 days. Twenty-one days post-vaccination, animal titers were increased 7-fold over controls. Forty-one days post-challenge, NO_2-treated animals exhibited an 11-fold enhancement. Even after 266 days of NO_2 exposure, titers were higher when compared to controls. Again, the authors hypothesized that NO_2 enhanced the ability of the monkey-adapted virus to become established and multiply.

Antweiler et al. (1975) did not find any alteration in guinea pig specific antibody titers when compared to controls, even after 33 days of exposure to 10,000 μg/m^3 (5.3 ppm) NO_2.

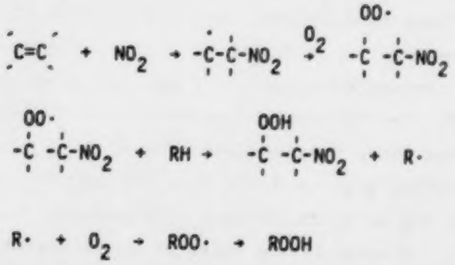
On the basis of experiments in which the continuous exposure of guinea pigs to 1,880 μg/m^3 (1 ppm) NO_2 for 6 months resulted in an increased incidence of infection, particularly within the lung, Kosmider and colleagues postulated an adverse effect of NO_2 on immune function (Kosmider et al., 1973b). These investigators also claimed that NO_2 causes decreases in complement concentrations when measured by a hemolysis assay; reductions in all immunoglobulin fractions when tested by immunoelectrophoresis; and increased mortality in mice exposed to 1,880 μg/m^3 (1 ppm) of NO_2 when infected intranasally with *D. pneumoniae*. Because of the importance of these observations, they require confirmation.

Balchum et al. (1965) exposed guinea pigs to 9,400 μg/m^3 (5 ppm) NO_2 for 4 hours/day, 5 days/week and to 9,400 μg/m^3 (5 ppm) NO_2 or to 28,200 μg/m^3 (15 ppm) NO_2 for 7-1/2 hours/day, 5 days/week. There was a noticeable increase in the titer of serum antibodies against lung tissue in all guinea pigs exposed to 9,400 μg/m^3 (5 ppm) or 28,200 μg/m^3 (15 ppm) NO_2 as early as 160 hours after NO_2 inhalation. The antibody titers increased with the intensity and duration of exposure to NO_2.

14.2.3.2 Lung biochemistry

14.2.3.2.1 Introduction. Nitrogen dioxide-related studies of lung biochemistry have been directed to either an investigation of the mechanism of toxic action of NO_2 or to the detection of indicators of early damage by NO_2 exposure. Two theories of action of NO_2 on biological systems have evolved as a result of these studies. The dominant theory is that NO_2 initiates lipid peroxidation, which subsequently causes cell injury or death and the symptoms associated with NO_2 inhalation. The second theory is that NO_2 oxidizes low molecular weight reducing substances and proteins. This oxidation results in a metabolic dysfunction which evidences itself as the toxic symptom. Nitrogen dioxide may, in fact, act by both means and, as a consequence, may affect the intermediary metabolism of animals and thus, their growth and maturation. Several potential biochemical mechanisms related to detoxification of NO_2 or to responses to NO_2 intoxication have been proposed. The effects of NO_2 exposure on lung biochemistry will be discussed in this context. (See Table 14-8.)

14.2.3.2.2 Lipid and diet effects. The dietary background of animals affects their response to all types of toxicants. For the most part, diet effects concerning NO_2 have been neglected and are unreported in the literature. A significant body of evidence has evolved, however, to support the idea that lipids and vitamin E are the most important dietary components in determining the response of animals to NO_2 exposure. Roehm et al. (1971) studied the *in vitro* oxidation of unsaturated fatty acids by O_3 and NO_2. A common mechanism of action was suggested for these two oxidizing air pollutants. Both NO_2 and O_3 initiated the oxidation of unsaturated fatty acids through free radicals. Typically, an induction period was noted with either anhydrous thin films or aqueous emulsions of linolenic acid exposed to 2,800 µg/m^3 (1.5 ppm) NO_2. The addition of free radical scavenging agents such as vitamin E, butylated hydroxytoluene (BHT), or butylated hydroxyanisol (BHA) delayed the onset of oxidation *in vitro*. The rate of oxidation of linolenic acid in thin films was proportional to concentrations of NO_2 ranging from 940 to 10,200 µg/m^3 (0.5 to 5.4 ppm). Thin-layer chromatography of the oxidation products of linolenic acid showed a conversion to polar nitrogen-containing compounds and to peroxides. A proposed mechanism of formation of these products follows (Menzel, 1976):



$$>C=C< + NO_2 \rightarrow -\dot{C}-C-NO_2 \xrightarrow{O_2} -\overset{OO\cdot}{C}-C-NO_2$$

$$-\overset{OO\cdot}{C}-C-NO_2 + RH \rightarrow -\overset{OOH}{C}-C-NO_2 + R\cdot$$

$$R\cdot + O_2 \rightarrow ROO\cdot \rightarrow ROOH$$

Nitrohydroperoxides and fatty acid hydroperoxides are produced from the oxidation of unsaturated fatty acids by NO_2. Phenolic antioxidants prevent the autoxidation of unsaturated

TABLE 14-8. EFFECTS OF NO_2 ON LUNG BIOCHEMISTRY

NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Effect	Reference
94 940	0.05 0.5	8 hr/day, 122 days	Guinea Pig	No effect on total weight of phospholipid. Significant alterations ($p < 0.05$) in individual phospholipid classes.	Trzeciak et al., 1977
94 or 940+ equal amount ammonia	0.05 or 0.5+ equal amount ammonia				
376 3,760 35,720	0.2 2 19	3 hr	Rat	At 376 $\mu g/m^3$ (0.2 ppm) inhibition of conversion of prostaglandin E_2 (PGE_2) to its metabolite (15-keto PGE_2) 18 hr post exposure. No effect on uptake or efflux of PGE_2. At 3,760 and 35,720 $\mu g/m^3$ (2 and 19 ppm), no effect on uptake of PGE_2. Efflux altered 18 hr post-exposure. Conversion of PGE_2 to 15-keto PGE_2 inhibited 18 and 60 hr post-exposure.	Menzel, 1980
750 1,880 5,640 9,400	0.4 1.0 3.0 or 5.0	72 hr	Guinea Pig	No effect at 750 $\mu g/m^3$. Increase in lung lavage protein and lipid content in vitamin C depleted but not normal at 1,880 $\mu g/m^3$. (See Edema Section 4.2.3.6)	Selgrade et al., 1981
9,400	5.0	3 hr	Guinea Pig	Increased lung lavage protein and lipid content in vitamin C depleted guinea pigs after 18 hr post exposure. (See Edema Section 4.2.3.6)	Selgrade et al., 1981

TABLE 14-8. (continued)

NO$_2$ Concentration µg/m^3	ppm	Exposure	Species	Effect	Reference
750	0.4	72 hr or 1 wk	Guinea Pig	No mortality or effect on lung lavage fluid composition. (See Edema Section 4.2.3.6)	Selgrade et al., 1981
750	0.4	Continuous, 1 wk	Guinea pig	Increase in lung protein content of guinea pigs with an unquantified vitamin C deficiency, most likely due to plasma leakage (see edema section 4.2.3.6). Some may result from cell death.	Sherwin and Carlson, 1973
750	0.4	4 hr/day, 7 days	Guinea pig	Increase in acid phosphatase (EC 3.1.3.2).	Sherwin et al., 1974
750 to 940	0.4 to 0.5	Continuous 1.5 yr	Mouse	Growth reduced; vitamin E (30 or 300 mg/kg diet) improved growth.	Csallany, 1975
1,790 to 1,880	0.95 to 1				
940	0.5	8 hr/day, 7 days	Guinea pig	Increase in serum LDH, CPK, SGOT, SGPT, plasma cholinesterase, lung and plasma lysozyme. Decrease in RBC GSH peroxidase. Lung GSH peroxidase and acid phosphatase unchanged.	Menzel et al., 1977
		8 hr/day, 4 mo		Decrease in plasma cholinesterase, plasma and lung lysozyme, and RBC GSH peroxidase. Lung GSH peroxidase unchanged. Increase in lung acid phosphatase.	

TABLE 14-8. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
940 1,880	0.5 1	Continuous, 17 mo	Mouse	Decreased body weight with vitamin E deficient, vitamin E supplemented (30 and 300 ppm) and DPPD supplemented (30 ppm). No change in tissue weight with exception of increased kidney weight in 1 ppm exposed animals with vitamin E deficient diet. Slightly decreased survival rate.	Csallany and Ayaz, 1978
940	0.5	Continuous, 17 mo	Mouse	No change in blood and lung GSH-peroxidase activity.	Ayaz and Csallany, 1978
1,880	1			Suppression of GSH-peroxidase activity.	
940 1,880	0.5 1	Continuous, 17 mo	Mouse	No increase in lipofuscin or glutathione peroxidase. Vitamin E (30 or 300 mg/kg) prevented lipofuscin accumulation (EC 1.11.19).	Ayaz and Csallany, 1977
1,880	1	Continuous, 2 wk	Rabbit	Decrease in lecithin synthesis after 1 wk; Less marked depression after 2 wk.	Seto et al., 1975
1,880 4,330 11,560	1 2.3 6.2	Continuous, 4 days	Rat	Activities of GSH reductase (EC 1.6.4.2) and glucose-6-phosphate dehydrogenase (EC 1.1.1.49) increased at 11,560 µg/m³ (6.2 ppm) level proportional to duration of exposure. Plasma lysozyme (EC 3.2.1.16) and GSH peroxidase (EC 1.11.1.9) not affected. No effects at 1,880 or 4,330 µg/m³ (1 or 2.3 ppm).	Chow et al., 1974
3,760	2	Continuous 1 to 3 wk	Guinea pig	Increase in number of lactic acid dehydrogenase (EC 1.1.2.3) positive cells with time exposure. Suggest Type I (LDH negative) cells decrease as Type II (LDH positive) cells increase.	Sherwin et al., 1972

TABLE 14-8. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
5,450	2.9	Continuous, 5 days/wk, 9 mo	Rat	Increase in lung wet weight (12.7%) and decrease in total lipid (8.7%). Decrease in saturated fatty acid content of lung lavage fluid and tissue. Increase in surface tension of lung lavage fluid.	Arner and Rhoades, 1973
5,640	3	Continuous, 17 days	Rat	Decrease in linoleic and linolenic acid of lavage fluid.	Menzel et al., 1972
18,800	10	Continuous 4 wk		Decrease in unsaturated fatty acids in lavage and lung tissue. Vitamin E (as d,l-α-tocopheryl acetate 100 mg/kg diet) reduced NO_2 effect.	
5,640	3	4 hr/day, 4 days	Squirrel monkey	Thickening of collagen fibrils.	Bils, 1976
5,600	3	7 days	Rat	No effects on parameters tested.	Mustafa et al., 1979a, b
13,200	7	4 days		Increase in lung weight, G-6-PD, glutathione reductase, glutathione peroxidase.	
18,800	10	4 days		Increase in lung weight, G-6-PD, 6-P-gluconate dehydrogenase, glutathione reductase.	
28,200	15	4 days		Increase in lung weight, DNA content, G-6-PD, 6-P-gluconate dehydrogenase, glutathione reductase, disulfide reductase, glutathione peroxidase, succinate oxidase, cytochrome oxidase; no effect on lung protein.	

TABLE 14-8. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
9,400	5	14 to 72 hr	Mouse	Increase in lung protein (14 to 58 hr) by radio-label.	Csallany, 1975
9,400	5	12 hr	Rat	Incorporation of ^{14}C-proline into insoluble collagen increased (58%).	Hackner et al., 1976
9,400	5	0.33, 1, 2 and 4 days	Rat	Increase in glucose utilization and lactate production. Lesser increase in pyruvate production.	Ospital et al., 1976
9,400 37,600 94,000	5 20 50	3 hr	Rabbit	Benzo(a)pyrene hydroxylase (EC 1.14.12.3) activity or tracheal mucosa not affected.	Palmer et al., 1972
11,000	6	4 hr/day, 30 days	Mouse	Increase in GSH reductase (EC 1.6.4.2) and glucose-6-phosphate dehydrogenase (EC 1.1.1.49) activities.	Csallany, 1975
15,000	8	Continuous 14 days	Mouse	Increase in lung protein.	Csallany, 1975
18,800	10	5 hr once a week 4 to 8 wk	Hamster (Vitamin A deficient)	Lipid droplets in alveolar walls. Alveolar necrosis and thickening of epithelial basement membrane with calcium deposits on inner and outer surfaces. Presence of virus particles within epithelial plasma membrane. Reduced DNA uptake. Decrease in basal cell growth. No reversion of Type II from Type I cell. No LDH isoenzyme III in terminal airway. Following 8 weeks of exposure, hypertrophy and hyperplasia of bronchiolar-epithelial cells, diffuse loss of cilia, membrane damage, and mitochondrial damage.	Kim, 1977; 1978

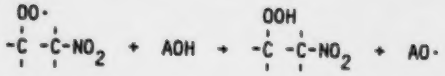
TABLE 14-8. (continued)

NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Effect	Reference
28,000 53,000	15 28	7 days	Mouse	Increase in GSH levels, GSH reductase, glucose-6-phosphate dehydrogenase, and GSH peroxidase.	Csallany, 1975
56,400	30	Continuous to 30 days	Hamster	Increase in lung proteolytic activity and in serum antiprotease at 2 days. Declined to normal values at 50 days.	Kleinerman and Rynbrandt, 1976
56,400	30	Continuous to 50 days	Hamster	Increase in lung proteolytic activity at 2 and 5 days, but the optimum pH was acidic (3.0). Not active at physiological pH of 7.2. Attributed to cathepsins, A, B_1, B_2, C, D, and E.	Rynbrandt and Kleinerman, 1977
56,400	30	22 hr/day, 3 wk	Hamster	Loss of body weight; increase dry lung weight; decrease in total lung collagen within 4 days and total lung elastin within 10 days. Collagen levels return to normal by day 14 of exposure. 3 wk following exposure, lung elastin levels had returned to normal.	Kleinerman and Ip, 1979
62,040	33	Continuous to 23 days	Rat	High level of dietary antioxidants increased the time until 50% mortality occurred (LT50). LT50 of vitamin E depleted rats was 11.1 days versus 170 days for vitamin E supplemented rats.	Menzel et al., 1972
75,200	40	5 hr	Rat	Increased incorporation of ^{14}C-palmitic acid in lung lecithin. Accumulation of disaturated lecithin by 6 hr post-NO_2 with maximum accumulation by 48 hr.	Blank et al., 1978

TABLE 14-8. (continued)

NO_2 Concentration					
μg/m³	ppm	Exposure	Species	Effect	Reference
75,200 132,000	40 70	2 hr	Rat	Benzo(a)pyrene hydroxylase (EC 1.14.12.3), phenol-O-methyl transferase (EC 2.1.1.25) and catechol-O-methyl transferase (EC 2.1.1.6) not affected.	Law et al., 1975

fatty acids by NO_2 by reacting with both fatty acid hydroperoxyl free radicals and nitrohydroperoxyl free radicals generated by addition of NO_2 to unsaturated fatty acids:



$$\overset{\mathrm{OO\cdot}}{-\mathrm{C}}-\mathrm{C}-\mathrm{NO_2} + \mathrm{AOH} \rightarrow \overset{\mathrm{OOH}}{-\mathrm{C}}-\mathrm{C}-\mathrm{NO_2} + \mathrm{AO\cdot}$$

$$\mathrm{ROO\cdot} + \mathrm{AOH} \rightarrow \mathrm{ROOH} + \mathrm{AO\cdot}$$

where AOH represents a phenolic antioxidant.

Rats evidenced increased mortality (Fletcher and Tappel, 1973; Menzel et al., 1972) and decreased content of unsaturated fatty acids in lung lavage fluid (Menzel et al., 1972; Thomas et al., 1968) when exposed to NO_2 concentrations ranging from 18,800 to 62,000 $\mu g/m^3$ (10 to 33 ppm). The effect was greater in animals fed diets depleted in vitamin E.

The effect of NO_2 exposure on the metabolism of vasoactive compounds by the rat lung was studied by Menzel (1980). Rats were exposed for 3 hours to 376 $\mu g/m^3$ (0.2 ppm), 3,760 $\mu g/m^3$ (2 ppm), and 35,720 $\mu g/m^3$ (19 ppm) NO_2 and their lungs were excised and perfused with ^3H-Prostaglandin E_2 (^3H-PGE_2), a natural product of the lung that acts on smooth muscles, up to 6 days at various times. NO_2 exposure did not affect the unidirectional uptake of ^3H-PGE_2, at 0 or 18 hr post-exposure, while efflux of ^3H-PGE_2 and its metabolites from the lung were altered 18 hours post exposure to 3,760 and 35,720 $\mu g/m^3$ (2 and 19 ppm) NO_2. Eighteen hours following 376, 3,760, and 35,720 $\mu g/m^3$ (0.2, 2, and 19 ppm) NO_2, the conversion of the perfused PGE_2 to its 15-keto metabolite was inhibited by 37, 41, and 62 percent, respectively. Recovery was not complete until 60 hr following 376 $\mu g/m^3$ (0.2 ppm) NO_2 exposure, and 90 hr following 3,760 $\mu g/m^3$ (2 ppm) exposure. Recovery had not occurred in animals exposed to 35,720 $\mu g/m^3$ (19 ppm) NO_2 for a period of 160 hours post exposure. No edema was observed following exposure to any of the three levels of pollutant.

Arner and Rhoades (1973) exposed rats to 5,450 $\mu g/m^3$ (2.9 ppm) NO_2 for 24 hours/day, 5 days/week, for 9 months. The lung wet weight increased by 12.7 percent compared to that of their control counterparts. The increase ($p < 0.05$) in lung wet weight was the same as the increase in lung water content. The lipid content of the lung was depressed ($p < 0.05$) by 8.7 percent. An analysis of the lungs showed that a decrease occurred in the total saturated fatty acid content. Unfortunately, values for unsaturated fatty acids of biological importance, such as the essential fatty acid arachidonic acid, were not reported. The surface tension of extracts of the lung increased, and the authors suggest that the increased surface tension corresponded to a decrease in the lung surfactant concentration.

Trzeciak et al. (1977) exposed guinea pigs to 940 $\mu g/m^3$ (0.5 ppm), 94 $\mu g/m^3$ (0.05 ppm), or these same NO_2 concentrations plus an equal amount of ammonia, for 8 hours/day for a total of 122 days. Lung phospholipids were analyzed, and no difference was found in the total weight of phospholipid of exposed versus control lungs. Significant alterations ($p < 0.05$) were found in the individual phospholipid classes. Decreases were noted in phosphatidyl ethanolamine, sphingomyelin, phosphatidyl serine, phosphatidyl glycerol-3-phosphate, and phosphatidic acid. Increases were noted in the lysophosphatidyl ethanolamine content, while the phosphatidyl choline (lecithin) content remained constant or was slightly depressed. Such changes could be indicative of change in cell type or cell function.

Lecithin synthesis appeared to be depressed in the lungs of rabbits exposed to 1,880 μm^3 (1 ppm) NO_2 for 2 weeks (Seto et al., 1975). The most marked effect was observed after 1 week of exposure and appeared to decline after the second week of exposure.

Csallany (1975) exposed mice continuously for 1.5 years to 750 to 940 μg/m^3 (0.4 to 0.5 ppm) or 1,790 to 1,880 μg/m^3 (0.95 to 1 ppm) NO_2 and fed the animals a basal diet which was either deficient in or supplemented with vitamin E at 30 or 300 mg/kg of diet. The author indicated that NO_2 reduced the growth rate in all four diet groups, but the vitamin E-supplemented groups were improved over the non-supplemented groups. High levels of vitamin E in the diet failed to provide greater improvement in growth rate over that of normal amounts of vitamin E in the diet. In other studies from this group, (Ayaz and Csallany, 1977; Ayaz and Csallany, 1978; Csallany and Ayaz, 1978) female weanling mice were exposed to 940 or 1,880 μg/m^3 (0.5 or 1 ppm) NO_2 continuously for 17 months. Animals were divided into three groups receiving the basal diet with either a normal supplement of vitamin E (30 mg/kg) or 300 mg/kg and a third group supplemented with the synthetic antioxidant N,N'-diphenylphenylenediamine (DPPD) at 30 mg/kg. After 17 months of exposure, the presence of lipofuscin pigment in the liver, lungs, spleen, heart, brain, kidney, and uterus was determined. While no effect could be ascribed to NO_2 exposure, vitamin E supplementation decreased the concentration of lipofuscin pigment in the liver, but not in other tissues. Lipofuscin pigment is proposed to be an end product of lipid oxidation accumulated in tissues.

Exposure of vitamin A deficient hamsters to 18,800 μg/m^3 (10 ppm) NO_2, 5 hours once a week for 4 to 8 weeks caused lung damage as compared to NO_2-exposed, non-vitamin A deficient hamsters (Table 14-8) (Kim, 1977; 1978).

A recent series of experiments have investigated the effect of vitamin C deficiency on NO_2 toxicity (Selgrade et al., 1981). Normal or vitamin C depleted guinea pigs were exposed to 752, 1880, 5460, or 9400 μg (0.4, 1.0, 3.0, or 5.0 ppm) NO_2/m^3 for 72 hrs and the lung lavage protein and lipid content determined. No effect was observed in guinea pigs having normal vitamin C blood contents, but depleted guinea pigs, having an average of 25% of the normal blood vitamin C content, had 2-5 times the control lavage fluid and lipid content with the exception of those guinea pigs exposed to 752 μg (0.4 ppm) NO_2/m^3. Exposure of normal or vitamin C depleted guinea pigs exposed to 752 μg/m^3 (0.4 ppm) for as long as 1 week had no effect on the composition of the lavage fluid. At 9400 μg (5 ppm) NO_2/m^3 for 72 hrs these changes in lavage fluid composition were correlated with mortality (50%) and alveolar edema as observed by conventional light microscopic histopathology in vitamin C depleted guinea pigs. When vitamin C depleted guinea pigs were exposed to 9400 μg (5 ppm) NO_2/m^3 for 3 hrs, increased protein and lipid contents were not observed until 15 hrs after exposure. These results conflict with those of Sherwin and Carlson who found increased protein content of lavage fluid from guinea pigs exposed to 752 µg/m^3 (0.4 ppm) for 1 week. Differences may be due to the reproducibility of exposure, methods of monitoring NO_2 during exposure, protein measurement in the lavage fluid and differences in the degree of vitamin C deficiency.

14.2.3.2.3 Sulfhydryl compounds and pyridine nucleotides. Oxidation of sulfhydryl compounds and pyridine nucleotides in the lung is well-established for O_3 exposures, (Evans et al., 1974) but little evidence has been reported for NO_2.

In experiments involving exposure of mice to very high (>143,000 µg/m^3; 76 ppm) concentrations of NO_2, several investigators reported that a wide variety of sulfur-containing compounds reduced the toxicity of NO_2 (Fairchild et al., 1959; Fairchild and Graham, 1963). For example when mice were first exposed to benzenethiol (14 ppm) for 24 to 72 hours prior to 4 hours of NO_2 exposure only 1/20 died, whereas 10/20 of the NO_2-exposed mice not pretreated with benzenethiol died. Inferences drawn from the protective effect of these compounds suggest that sulfhydryl compounds within the lung were being oxidized to disulfides (Fairchild et al., 1959). Included among the compounds observed to exert a protective effect are: (1) hydrogen sulfide (2) benzenethiol, (3) d,α-napthylurea, (4) phenylthiourea, and (5) a number of thyroid-blocking agents.

Ospital et al. (1976) reported that exposure to 9,400 µg/m^3 (5 ppm) NO_2 for 8 hours altered the glucose metabolism of slices made from the lungs of exposed rats. Glucose utilization and lactate production were increased by 28 and 43 percent, respectively, while pyruvate production rose by 6 percent. Exposure of rats to 9,400 µg/m^3 (5 ppm) NO_2 for 1, 2, and 4 days produced similar alterations, but individual values were not reported. Neither increased hexose monophosphate shunt nor citric acid (Krebs) cycle activity could account for the increased glucose utilization. The authors concluded that NO_2 exposure increased the activity of the glycolytic pathway and suggested that this increase may be related to an increased biosynthesis due to injury.

14.2.3.2.4 Effects on lung amino acids, proteins, and enzymes. Concentrations of NO_2 >9,400 µg/m^3 (5 ppm) produce lung edema with concommitant infiltration of serum protein and enzymes. Alterations in the cell types of the lung also occur (see Section 14.2.3.3). Thus, some reports of changes in lung enzymes and proteins may reflect either edema or altered cell populations rather than direct effects of NO_2 on lung enzymes.

Sherwin et al. (1972) exposed guinea pigs to 3,760 µg/m^3 (2 ppm) NO_2 for 1, 2, or weeks. They examined lung sections histochemically for lactic acid dehydrogenase (LDH). With this technique, LDH is primarily an indicator of Type II pneumocytes rather than Type I. The number of Type II pneumocytes per alveolus was determined. In control lung sections, a mean of 1.9 Type II cells per alveolus was found with a range of 1.5 to 3.4 Type II cells per alveolus in upper lobes of the lung. A range of 1.6 to 3.1 Type II pneumocytes per alveolus was found in the lower lobes. Exposure to NO_2 increased the LDH content of the lower lobes of the lung by increasing the number of Type II cells per alveolus ($p < 0.05$). The increase was progressive over the 1-, 2-, and 3-week exposure period. The authors then contended that the increase in lung LDH content was due to the replacement of Type I pneumocytes by Type II pneumocytes as shown in morphological studies (see Section 14.2.3.3).

Several other biochemical indicators of lung damage have been studied. Sherwin et al. (1974) exposed guinea pigs with an unquantified vitamin C deficiency to 750 μg/m^3 (0.4 ppm) NO_2 for 4 hours/day for 7 days and found an increase in acid phosphatase activity ($p < 0.05$). An increased aldolase activity was reported by Kosmider et al. (1975) in the blood, liver, and brain of NO_2-exposed guinea pigs, but was statistically significant only in liver samples. Values for the lung and exposure levels were not reported.

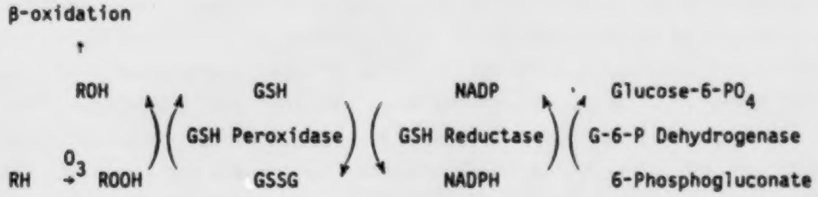
The effect of NO_2 on the important enzyme benzpyrene hydroxylase was studied by Palmer et al. (1972). Since lung cancer in man is predominantly of bronchial rather than parenchymal origin, benzpyrene hydroxylase activity of the tracheobronchial region of the lung was studied in rabbits which had been exposed to 9,400, 37,600, or 94,000 μg/m^3 (5, 20, or 50 ppm) NO_2 for 3 hours. No effect was observed on the benzpyrene hydroxylase activities in NO_2 exposure, but O_3 exposure of 1,400 to 19,600 μg/m^3 (0.75 to 10 ppm) markedly decreased benzpyrene hydroxylase activity in a dose-related manner. Law et al. (1975) studied the effect of NO_2 on benzpyrene hydroxylase, microsomal O-methyl transferase, catechol O-methyl transferase, and supernatant catechol O-methyl transferase activities of the lungs of rats. While benzpyrene hydroxylase activity of the lung could be induced by treatment with the carcinogen, 3-methylcholanthrene, exposure to 75,200 or 132,000 μg/m^3 (40 or 70 ppm) NO_2 for 2 hours had no effect. Thus, the studies of Palmer et al. and Law et al. agree that NO_2 has no effect on benzpyrene hydroxylase activity of the lung. The O-methyl transferase activity studied by Law et al. relates to the ability of the lung to metabolize the important catecholamine hormones. This metabolism does not appear to be affected by NO_2 treatment.

Menzel et al. (1977) exposed guinea pigs 8 hr/day to 940 μg/m^3 (0.5 ppm) NO_2 for 4 months. After an initial exposure of 7 days, serum LDH, total creatine phosphokinase (CPK), glutamic-oxalacetic transaminase (SGOT), and glutamic-pyruvic transaminase (SGPT) were elevated. Lung GSH peroxidase and acid phosphatase were not affected. In contrast to the findings of Chow et al., (1974) lung lysozyme levels were elevated as were plasma levels. The release of isomeric forms of CPK was characteristic of generalized damage to the lung. The elevation in total CPK was statistically significant ($p < 0.05$) while the elevations in LDH, SGOT, and SGPT were not significant because of the large variance in the exposed groups.

A major concern has been the effect of NO_2 exposure on the structural proteins of the lung, since elastic recoil is lost following exposure. Bils (1976) reported a thickening of the collagen fibrils in squirrel monkeys exposed to 5,640 μg/m^3 (3 ppm) NO_2 for 4 hours/day for 4 days. Kosmider et al. (1973a) reported that the urinary hydroxyproline and acid mucopolysaccharide contents of guinea pigs exposed to 1,880 μg/m^3 (1 ppm) NO_2 for 6 months were increased ($p < 0.05$). Presumably these increases represented degradation of collagen. Hacker et al. (1976) measured the incorporation of ^{14}C-proline into soluble and insoluble collagen fractions in the lungs of rats exposed to 9,400 μg/m^3 (5 ppm) NO_2 for 12 hours. Incorporation of ^{14}C-proline into insoluble collagen was 58 percent greater in the NO_2-exposed animals than in air-exposed control groups, supporting the biochemical evidence for greater collagen turnover in NO_2-exposed animals.

Enzymes observed to have increased activity following exposure to high concentrations of NO_2 included aldolase (in vitro) (Ramazzotto and Rappaport, 1971) and serum antiprotease (in vivo) (Kleinerman and Rynbrandt, 1976). Plasma lysozyme activity was reported to be unaffected (in vivo) (Chow et al., 1974).

14.2.3.2.5 Potential defense mechanisms. Menzel (1970; 1976) proposed that antioxidants might protect the lung from damage by NO_2 by inhibiting lipid peroxidation. Data related to this hypothesis have been reported. (Ayaz and Csallany, 1977; Ayaz and Csallany, 1978; Csallany, 1975; Fletcher and Tappel, 1973; Menzel et al., 1972; Thomas et al., 1968) Chow and Tappel (1972) proposed an enzymatic mechanism for the protection of the lung against lipid peroxidation damage by ozone. They proposed the following scheme:



where R is an aliphatic organic radical

Chow et al. (1974) exposed rats to 1,880, 4,330, or 11,560 µg/m^3 (1, 2.3, or 6.2 ppm) NO_2 continuously for 4 days to determine the effect on the glutathione peroxidase system. They determined the activity of GSH reductase, glucose-6-phosphate dehydrogenase, and GSH peroxidase in the soluble fraction of exposed rat lungs. Linear regression analysis of the correlation between the NO_2 concentration and enzymatic activity was found to have a significant positive correlation coefficient of 0.63 ($p < 0.001$) for GSH reductase and 0.84 ($p < 0.003$) for glucose-6-phosphate dehydrogenase. No correlation was found between the GSH peroxidase activity and the NO_2 exposure concentration. The activities of GSH reductase and glucose-6-phosphate dehydrogenase were significantly increased during exposure to 11,560 µg/m^3 (6.2 ppm) NO_2.

Ayaz and Csallany (1978) exposed female mice continuously for 17 months to 940 and 1,880 µg/m^3 (0.5 and 1 ppm) NO_2 and fed the animals a basal diet which was either deficient in vitamin E or supplemented with 30 or 300 mg/kg of diet. Blood, lung, and liver tissues were assayed for glutathione peroxidase activity. Exposure to 940 µg/m^3 (0.5 ppm) NO_2 did not alter blood or lung GSH peroxidase; however, 1,880 µg/m^3 (1 ppm) NO_2 exposure suppressed enzyme activity. A combination of vitamin E deficiency and 1,880 µg/m^3 (1 ppm) NO_2 exposure resulted in the lowest GSH peroxidase in blood and lung. Liver GSH-peroxidase was unaffected by either vitamin deficiency or NO_2 exposure.

Donovan et al. (1976) and Menzel et al. (1977) exposed guinea pigs continuously to 940 µg/m^3 (0.5 ppm) NO_2 for 4 months. After an initial short-term exposure of 7 days or at the completion of a long-term exposure at 4 months, animals were killed, and the lung and red blood cell (RBC) GSH peroxidase levels were determined. Short-term exposure to NO_2 depressed RBC GSH peroxidase but did not affect lung levels. Long-term exposure, on the other hand, affected neither lung nor RBC GSH peroxidase. These studies confirm the results in rats and indicate a distinct difference in the effect of NO_2 and O_3 on the lung.

Since protection against NO_2 occurs with vitamin E, lipid peroxidation most likely occurs, but the GSH peroxidase defense system does not appear to be induced. Chow et al. (1974) concluded: "Since exposure of rats to NO_2 has insignificant effect on lung GSH peroxidase activity, but had significantly increased the activities of GSH reductase and G-6-P dehydrogenase, it appears that this oxidant attacks mainly glutathione and NADPH while O_3 not only initiates lipid peroxidation but also directly attacks these reducing substances."

Selgrade et al. (1981) expanded the studies of Sherwin and Carlson (1972) on the effects of vitamin C deficiency on NO_2 toxicity. These studies were aimed at measuring the infiltration of plasma proteins into the airways as an index of NO_2-induced damage. The degree of vitamin C deficiency produced by Selgrade et al. was mild, being on the average a sufficient reduction in vitamin C intake to produce a 25% decrease of the vitamin C blood levels. At high levels of NO_2 exposure, 9400 µg (5.0 ppm) NO_2/m^3 for 72 hrs, 50% of the vitamin C depleted guinea pigs died, while none of the normally supplemented animals was affected. When exposed to greater than 752 µg/m^3 (0.4 ppm), lavage fluid protein and lipid content was increased. Unlike the studies of Sherwin and Carlson (1973), Selgrade et al. found no effect at 752 µg/m^3 (0.4 ppm) for up to 1 week. The vitamin C status of the guinea pigs in the Sherwin and Carlson study is not documented in sufficient detail to judge if this is the reason for the discrepancy in the two studies, but taken together these investigations support a role for dietary vitamin C as well as vitamin E in influencing the susceptibility of animals to NO_2. Since vitamin C is readily oxidized and reduced, it could serve to detoxify oxidative products formed by NO_2 or to maintain the intracellular redox potential.

14.2.3.3 Morphology Studies--Nitrogen dioxide produces morphological alterations starting in the terminal airways and adjacent alveoli. (See Table 14-9.) A comprehensive summary review has been prepared by Coffin and Stokinger (1977).

The events leading to emphysema from NO_2 exposure of the rat have been described by Freeman and co-workers. (Cabral-Anderson et al., 1977; Evans et al., 1972; 1973a; 1973b; 1974; 1975; 1976; 1977; Freeman et al., 1966; 1968c; 1972; Stephens et al., 1971; 1972) The earliest alterations resulting from exposures to concentrations above 22,600 µg/m^3 (12 ppm) were seen within 24 hours of continuous exposure. These alterations included increased macrophage aggregation, desquamation of the Type I pneumocytes and ciliated bronchiolar cells, and accumulation of fibrin in the small airways. The cuboidal Type II pneumocytes slowly differentiate into the squamous Type I cells as replacements and alter the appearance of the parenchyma into a "gland-like" tissue in the region of the ducts. Incorporation of ^{3}H-thymidine by Type II

TABLE 14-9. EFFECT OF NO_2 ON LUNG MORPHOLOGY

NO_2 Concentration		Exposure	Species	Effect	Reference
µg/m³	ppm				
188 + daily 2-hr spike of 1,880	0.1 + daily spike of 1	Continuous, 6 mo	Various species	Emphysematous alterations.	Port et al., 1977
470	0.25	4 hr/day, 5 days/wk, 24 or 36 days	Rabbit	Isolated swollen collagen fibers.	Buell, 1970
553	0.34	6 hrs/day, 5 days/wk 6 wk	Mice	Hyperplasia and hypertrophy of alveolar type II cells. Decrease in body weight, spleen weight, and area of splenic lymphoid nodules.	Sherwin et al., 1979 Kuraitis et al., 1979
940	0.5	Continuous, 12 mo	Mouse	At 10 days: Clara cell damage Loss and shortening of cilia Alveolar edema in interstitial space and epithelium At 35 to 40 days: Bronchial hyperplasia At 6 mo: Fibrosis At 12 mo: Bronchial hyperplasia	Hattori, 1973 Hattori and Takemura, 1974
940	0.5	6, 18, or 24 hr/day, to 12 mo	Mouse	Alveolar damage. Interstitial pneumonia may have confused interpretation.	Blair et al., 1969
940 1,880	0.5 1	4 hr 1 hr	Rat	Degranulation of mast cells. Response seemed reversible.	Thomas et al., 1967
940 to 1,500	0.5 to 0.8	Continuous, 1 mo	Mouse	Damage to tracheal mucosa and cilia.	Hattori et al., 1972 Nakajima et al., 1969
1,030 to 3,000	0.55 to 1.6	Continuous, 5 wk	Mouse	Damaged cilia, increase in mucus secretion by nonciliated cells.	Miyoshi, 1973

TABLE 14-9. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
1,500	0.8	Continuous, to 33 mo	Rat	Normal growth. Decreased respiratory rate (~20%). Tachypnea exaggerated with exposure. Normal gross and microscopic appearance. Suggestive evidence of changes in terminal bronchioles.	Freeman et al., 1966
1,880	1	Continuous, 493 days	Monkey	Virus-challenged animals had slight emphysema, thickened bronchial and bronchiolar epithelium. NO_2 exposure alone produced no effect.	Fenters et al., 1973
1,880 to 2,820	1 to 1.5	Continuous, 1 mo	Mouse	Same morphology as others. Recovery for 1 to 3 mo showed lymphocyte infiltration up to 3 mo.	Chen et al., 1972
3,760	2	Continuous, 43 days	Rat	No changes in terminal bronchi. Cilia lost and altered by 72 hr. Greater cilia loss and focal hyperplasia by 7 days. Regeneration of cilia by 14 days. Substantial recovery by 21 days.	Stephens et al., 1972
32,000	17			Earlier and greater injury of same type and sequence as at lower level with loss of Type I cells.	
3,760	2	Continuous, 3 wk	Guinea pig	Increased number of LDH positive cells/ alveolus (presumably Type II cells)	Sherwin et al., 1972
3,760	2	Continuous, 3 wk	Guinea pig	Type II cell hypertrophy.	Sherwin et al., 1973
3,800	2	Continuous, 14 mo	Monkey (Macaca speciosa)	Hypertrophic bronchiolar epithelium, particularly in the area of respiratory bronchiole. NaCl aerosol had no added effect.	Furiosi et al., 1973
3,760	2	Continuous, to 360 days	Rat	At 3,760 µg/m^3 (2 ppm), cell division of Type II cells peripheral to terminal brochiolar-alveolar junction seen.	Evans et al., 1972; 1978a
32,000	17	Continuous, 7 days		Cell division seen at 24 hr, peak at 2 days, decreased to preexposure by 5 days.	

TABLE 14-9. (continued)

NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Effect	Reference
32,000	17	Continuous, 1 day		Type II cell proliferation.	
5,640	3	4 hr/day, 4 days with intermittent exercise	Squirrel monkey	Thickening of alveolar wall and basal lamina. Interstitial collagen.	Bils, 1976
9,400 18,800	5 10	Continuous, 90 days	Monkey	Infiltration of macrophages, lymphocytes, and occasionally polymorphonuclear leukocytes. Hyperplasia of bronchiolar epithelium and Type II cells.	Busey et al., 1974
18,800	10	Continuous, 90 days	Rat	Decrease in length and weight of neonates exposed, delivered and reared in NO_2.	Freeman et al., 1974b
28,200	15	Continuous, 75 days		Delayed lung development in progeny exposed *in utero* and raised in NO_2. 75 days required to make up deficit.	
18,800	10	Continuous, 6 wk	Guinea pig	Type II cell hypertrophy, 1 to 6 wk exposure with increased lamellar bodies within Type II cells.	Yuen and Sherwin, 1971
18,800 to 47,000	10 to 25	6 mo	Dog	Emphysema and death.	Riddick et al., 1968
19,100 to 21,500	10.2 to 11.4	12 mo	Cat	Intraluminal mucus. Increase in goblet cells. Thickening of epithelium. Fly ash (9,950 to 10,200 $\mu g/m^3$) had no effect.	Kleinerman et al., 1976

TABLE 14-9. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
28,000	14	48 hrs	Rat	Neonate-20 days of age: loss of cilia; flat luminal surface of epithelium. No nodules. No significant injury of Type I cells. Weanling-30 to 35 days of age: tissue nodules (size increases with exposure), Type I cell injury. 40 days: hypertrophy and stratification of nonciliated cells in terminal bronchioles; polyploid extensions of epithelium in terminal airways.	Stephens et al., 1978
28,000 to 32,000	15 to 17	24 hr	Rat	Division of Clara cells replaced damaged ciliated cells.	Evans et al., 1976
28,200	15	48 hr	Rat	Increased cell division, especially Type II cells.	Evans et al., 1974
28,200	15	4 days/wk, 5 wk, total 31.5 hr	Rat	No effect on blood methemoglobin.	Csallany and Ayaz, 1978
		5 days/wk, 18 wk, total 93.5 hr		In lung increased atelectasis and alveolar thickening. In liver increased granular changes, karyolysis and karyorhexis. Suppressed by Vitamin E supplementation. No effect on tissue lipofuscin pigment by NO_2 exposure or dietary Vitamin E. Lung lipid extract distribution not affected by NO_2 exposure.	
28,000	15	Continuous, 1,4,10,16 and 20 wk	Rat	Hyperplasia of terminal bronchiolar and alveolar epithelium reversible on discontinuation of exposure; alterations of interstitial structural features of alveoli not reversible.	Freeman et al., 1969
~28,000	15	Subacute	Rat	Newborn rats up to age 3 wk relatively resistent to exposure compared to mature rats.	Lunan et al., 1977
28,200	15	24 hr	Hamster	^{3}H-thymidine (Tdr) 24 hr post-NO_2, 1 and 24 hr post TdR, label in smallest ciliated airways, less in trachea. Increase in lung parenchyma distant from airways. Deep parenchyma 8% Type II cells of which 11% labelled 24 hr post-NO_2. 25% AM labelled. No change in AM population.	Hackett, 1978

TABLE 14-9. (continued)

NO_2 Concentration μg/m³	ppm	Exposure	Species	Effect	Reference
28,200 to 32,000	15 to 17	48 hr	Rat	Alveolar macrophage division seen with DNA synthesis.	Evans et al., 1973a
28,200 to 32,000	15 to 17	Continuous, lifetime	Rat	Bronchial epithelium hypertrophic and metaplastic. Increased mucus. Connective tissue damage. Fibrosis at junction of respiratory bronchiole and alveoli. Emphysema.	Freeman et al., 1968c; 1972
28,000 to 31,960	15 to 17	12 and 24 hr	Rat	Loss of cytoplasmic projections of nonciliated (Clara) cells and exfoliation of ciliated cells.	Stephens et al., 1971 Evans et al., 1978b
32,000	17	Continuous, 90 days	Rat	Collagen damage. Large fibers and thickened basement membrane.	Stephens et al., 1971
69,000	37.2	4 hr	Dog	Interstitial edema.	Guidotti and Liebow, 1977
150,000	80	3 hr	Cat	Some alveolar desquamation. 12 and 24 hr post-NO_2, degeneration of Clara cells, loss of cilia distal portion of terminal bronchioles. Clara cell hyperplasia evident 48 to 168 hr post-NO_2. Loss of pneumonocytes resulted in substantial centroacinar denudation of basal lamina in lungs 12 and 24 hr post-NO_2. Serous and serofibrinous edema, neutrophilic emigration and extravasation of erythrocytes evident 24-hr post-NO_2 exposure. Peribronchiolar congestion and edema. 50% proximal alveolar spaces filled with serous edema fluid. Increased number of AM and hyperplasia of type II pneumocytes.	Langloss et al., 1977

cells was observed within 12 hours after initial exposure, the number of labeled cells becoming maximal in about 48 hours and decreasing to pre-exposure levels by 6 days, despite persistent exposure (Evans et al., 1975). This pattern of incorporation of ^{3}H-thymidine, indicative of cell replication, was documented at 3,760 μg/m^3 (2 ppm) NO_2 as well as at 32,000 μg/m^3 (17 ppm) (Evans et al., 1972). On continued exposure, there is a change in the normally irregular contour formed by the ciliated and nonciliated cuboidal cell layer of the terminal airways due to a loss of the bud-like cytoplasmic projections of nonciliated (Clara) cells and the exfoliation of ciliated cells (Evans et al., 1978b). Later, aberrations in ciliogenesis occur and cilia often appear within vacuoles surrounded by cytoplasm.

Fenters et al. (1973) exposed monkeys to 1880 μg/m^3 (1 ppm) NO_2 continuously for 493 days. Four monkeys were challenged with influenza A/PR/8/34 virus one day before and 41, 83, 146, and 266 days after initiation of NO_2 exposure. Monkeys exposed to NO_2 and virus developed moderate emphysema with thickened bronchial and bronchiolar epithelium. No effect was observed in monkeys exposed to NO_2 alone or controls.

Age is a factor in determining the response of the lungs to NO_2. Newborn rats up to the age of about 3 weeks are relatively resistant to high subacute concentrations (about 28,000 μg/m^3; 15 ppm) compared to more mature animals. (Lunan et al., 1977) On the contrary, old rats about 2 years of age or more have a 24-hour delay in renewing injured or desquamated Type I pneumocytes, relative to younger animals, and also have a lower threshold for death from pulmonary edema induced by NO_2 (Evans et al., 1977).

Sherwin and co-workers (Kuraitis et al., 1979; Sherwin et al., 1979) exposed mice to 553 μg/m^3 (0.34 ppm) NO_2 6 hours/day, 5 days/week for 6 weeks. Lactate dehydrogenase positive type II cells were quantitated and shown to be hyperplastic and hypertrophied. Body weights, spleen weights, and area of splenic lymphoid nodules were decreased following exposure to NO_2.

Buell (1970) reported the isolation of swollen, damaged, insoluble collagen fibers from the lungs of rabbits exposed to 470 μg/m^3 (0.25 ppm) for 4 hours/day, 5 days/week for 24 or 36 days. Modifications of collagenous tissue are evident early and late and may be reflected in the increased excretion of collagen degradation products in the urine.

While hyperplasia of the terminal bronchiolar and alveolar epithelium is reversible on discontinuation of exposure to 28,000 μg/m^3 (15 ppm) NO_2, the interstitial structural alterations of alveoli are not (Freeman et al., 1969a). Bronchiolar epithelial alterations are observed during lifetime exposure to 3,760 μg/m^3 (2 ppm) (Freeman et al., 1968a,c). Similar changes in rats without enlargement of lungs have been seen after a lifetime exposure to 1,500 μg/m^3 (0.8 ppm) NO_2 (Freeman et al., 1968c).

Embryonic and adult hamster tracheal cells were exposed as cultures to 1,880 μg/m^3 (1 ppm) NO_2 for 6 hours (Samuelson et al., 1978). Cells so treated lost their ability to grow and form colonies. Hamster lung fibroblasts (V-79), when exposed *in vitro* to 216 μg/m^3 (0.12 ppm) NO_2 for periods up to 6 hours, also failed to divide and form colonies.

Blair et al. (1969) did microscopic studies of the temporal alterations of lung morphology in mice exposed to 940 μg/m^3 (0.5 ppm) NO_2 for 6, 18, and 24 hours per day. Exposed mice were found to have expanded alveoli after 3 to 12 months of exposure. However, interstitial pneumonia may have confused the interpretation. Continuous exposures of mice to 940 to 1,500 $\mu g/m^3$ (0.5 to 0.8 ppm) for 1 month induced proliferation of epithelial cells of the mucous membranes, degeneration and loss of mucous membranes, edema in alveolar epithelial cells, loss of cilia, and an influx of monocytes. (Hattori, 1973; Hattori and Takemura, 1974; Hattori et al., 1972; Nakajima et al., 1969) Mice exposed continuously for 5 weeks to 1,030 to 3,000 $\mu g/m^3$ (0.55 to 1.6 ppm) NO_2 exhibited damaged cilia and an increase in mucus secretion by nonciliated cells.

Furiosi et al. (1973) investigated the influence of a 14-month continuous exposure to 3,800 $\mu g/m^3$ (2 ppm) NO_2 and 330 $\mu g/m^3$ (0.1 ppm) NaCl (0.1 to 10.3 μm), alone and in combination, on monkeys (Macaca speciosa). Rats were exposed simultaneously but received approximately 1,880 $\mu g/m^3$ (1 ppm) NO_2 due to differences in the exposure cages. The NaCl exposure alone caused no effects. In monkeys, the NO_2 exposure resulted in hypertrophy of the bronchiolar epithelium, particularly in the area of the respiratory bronchiole which is confluent with the alveolar duct. Morphological changes were observed also in the more proximal bronchiolar epithelium. When NaCl was combined with NO_2, NaCl appeared to have no influence. Neither were alveolar epithelial changes noted. In rats, the results were similar to an earlier study with equivocal findings, (Freeman et al., 1966) in which the animals were exposed to about 1,500 $\mu g/m^3$ (0.8 ppm) NO_2 for over 2 years. In the latter study, (Freeman et al. 1966) it was reported that rats exposed continuously up to 33 months exhibited an essentially normal gross and microscopic appearance with suggestive evidence of changes in the terminal bronchioles.

Recovery from exposure to NO_2 has been reported (Chen et al., 1972; Evans et al., 1978b). In mice sacrificed after exposure to 1,880 to 2,820 $\mu g/m^3$ (1 to 1.5 ppm) NO_2 for 30 days, the morphological changes were similar to those described above (Chen et al., 1972). Lymphocytes infiltrated around the bronchioles during the ensuing 1- to 3-month period in clean air. This was not observed in mice sacrificed either during or immediately after NO_2 exposure, leading the authors to speculate that this might have been an autoimmune response.

Bils (1976) observed connective tissue changes in squirrel monkeys that respired 5,640 $\mu g/m^3$ (3 ppm) NO_2 for 4 hours/day for 4 days with intermittent exercise during exposure. Thickening of the alveolar wall between the air and capillary spaces, in the basal lamina, and in the interstitial areas was seen. Numerous fenestrations were found in the alveolar walls in the centroacinar area.

Coffin and Stokinger (1977) suggest that fenestrations are related to the pores of Kohn. Since the frequency of such pores differs among species, attenuation of alveolar septae and distention of their pores may be recognized, depending on the species. Reduction in the elasticity of connective tissue, regardless of species, combined with the loss of Type I cells, could result in enlargement of the pores. They contend that the process may be largely irreversible once the pores are enlarged. Thus, the mechanisms resulting in their appearance, although fenestration may not be prominent in all species, may be a hallmark of pathogenesis.

Port et al. (1977) investigated the effects of NO_2 on several species using light and scanning electron microscopy. Exposure to 188 μg/m^3 (0.1 ppm) was continuous for 6 months. Upon this regimen were superimposed daily 2-hour peaks of 1,880 μg/m^3 (1 ppm) NO_2. Although bronchioles and alveolar ducts were not found to be remarkable, occasional foci of distended alveoli were seen under the pleura. Large variations in pore size (up to 5-fold) and in number (up to 10 per alveolus) were seen. Alveolar pores were thought to be involved in the development of emphysema induced by NO_2 in some species but not in others.

In exposures to 940 μg/m^3 (0.5 ppm) for 4 hours or 1,880 μg/m^3 (1 ppm) for 1 hour, rats sustained reversible lung-tissue change (Thomas et al., 1967). In tissues from animals sacrificed immediately after exposure, the mast cells were ruptured and disoriented and showed loss of cytoplasmic granules. The occurrence was primarily in the pleura, bronchi, and surrounding tissues, but most markedly in the mediastinum. This response seemed reversible, since animals sacrificed 24 to 27 hours after exposure appeared to have only a few ruptured mast cells. The investigators considered the release of granular material from the lung mast cells in response to NO_2 inhalation to signify the potential onset of an acute inflammatory reaction.

14.2.3.4 Pulmonary Function--Exposures of animals to 9,400 μg/m^3 (5 ppm) NO_2 or lower have been reported to have produced a variety of effects on pulmonary function. (See Table 14-10.) Elevated respiratory rates throughout the life-span of rats were observed after the animals were exposed to 1,500 μg/m^3 (0.8 ppm) NO_2 for periods up to 2.75 years (Freeman et al., 1966; Haydon et al., 1956).

Rats exposed to 5,400 μg/m^3 (2.9 ppm) NO_2 for 24 hours/day, 5 days/week, for 9 months exhibited a 13 percent ($p < 0.05$) decrease in lung compliance and lowered lung volumes when compared to controls (Arner and Rhoades, 1973). Freeman et al., (1968c) however, observed that resistance to airflow and dynamic compliance were not affected when rats were exposed for 2 years to 3,800 μg/m^3 (2 ppm) NO_2. Tachypnea (rapid breathing) was observed.

Rats and cats exhibited a tendency toward increased respiratory rates and decreased arterial O_2 partial pressure when exposed to 940 to 38,000 μg/m^3 (0.5 to 20 ppm) NO_2 (Zorn, 1975b). Oxygen uptake in the blood was impaired and remained so several hours after exposure was terminated (Zorn, 1975a).

Murphy et al. (1964) exposed guinea pigs to various concentrations of NO_2 between 9,780 and 24,440 μg/m^3 (5.2 and 13 ppm). At 9,780 μg/m^3 (5.2 ppm), there were no significant increases in respiratory rate until after 3 hours of exposure. When guinea pigs exposed to this concentration for 4 hours were returned to clean air, recovery occurred within approximately 1 hour. At higher concentrations, respiratory rates increased earlier. Tidal volume also decreased during the 4-hour exposure to 9,780 μg/m^3 (5.2 ppm). The net effect was to maintain minute ventilation at a nearly constant level. No significant alterations in respiratory function in rabbits were observed when animals were exposed to 9,400 μg/m^3 (5 ppm) NO_2 for 6 hours/day over a period of 18 months (Wagner et al., 1965).

TABLE 14-10. PULMONARY FUNCTIONS

Pollutant	NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Effect	Reference
NO_2	940 to 38,000	0.5 to 20	Continuous	Rat, cat	Increased respiratory rates. Decreased arterial oxygen pressure. Impaired O_2 uptake in blood.	Zorn, 1975a; 1975b
NO_2	1,500	0.8	Continuous, to 2.75 yr	Rat	Increased respiratory rates.	Freeman et al., 1966 Haydon et al., 1956
NO_2	1,880	1	16.5 mo. NO_2 alone or with subsequent influenza virus A/PR/8/34 challenge.	Squirrel monkey	Little change in tidal volume, minute volume, and respiratory rate.	Fenters et al., 1973
NO_2	3,800	2	2 yr	Rat	Resistance or dynamic compliance unchanged. Tachypnea.	Freeman et al., 1968c
NO_2	3,800 16,900	2 9	10 yr 5 yr	Monkey (Macaca speciosa), Pregnant and offspring	No change in mean respiratory rate or functional residual capacity of juveniles.	Freeman and Juhos, 1976
NO_2	5,400	2.9	Continuous, 5 days/wk, 9 mo	Rat	Decreased (13%, $p < 0.05$) lung compliance and lowered lung volume.	Arner and Rhoades, 1973
NO_2	9,400	5	6 hr/day, 18 mo	Rabbit	Respiratory function unchanged.	Wagner et al., 1965
NO_2	9,400	5	7.5 hr/day, 5 days/wk, 5.5 mo	Guinea pig	No increase in total resistance to airflow.	Balchum et al., 1965

TABLE 14-10. (continued)

Pollutant	NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
NO_2	9,400 18,800	5 10	Continuous, 90 days	Cynomologus monkey	Unchanged with heat stress. Impaired distribution of ventilation of the lungs; increased respiratory rates; decreased tidal volume; addition of heat stress decreased dynamic compliance of lungs.	Coate and Badger, 1974
NO_2	9,400	5	Continuous, 2 mo. *K. pneumoniae* challenge	Squirrel monkey	Gradual reduction in tidal volume. Increased respiratory rate. Minor changes in minute respiratory volume. After challenge, minute volumes decreased and remained depressed.	Henry et al., 1970
	18,800	10	Continuous, 1 mo. *K. pneumoniae* challenge		Elevation in minute respiratory volume due to increased tidal volume and respiratory rate by 2 wk and throughout exposure. 3 days after challenge, marked reduction in minute volume.	
NO_2	9,780	5.2	4 hr	Guinea pig	Increased respiratory rate, return to normal levels in clean air. Decreased tidal volume.	Murphy et al., 1964
NO_2 SO_2 NO_2 + SO_2	10,000 10,000 10,000 + 10,000	5.3 3.8 5.3 + 3.8	6 days/wk, 6 mo	Guinea pig	Respiratory frequency, flow rate, or minute volume unchanged in all regimens.	Antweiler and Brockhauser, 1976
NO_2	15,000 to 22,600	8 to 12	continuous, 12 wk	Rabbit	Increased nonelastic resistance and functional residual capacity. Static lung compliance unchanged.	Davidson et al., 1967

TABLE 14-10. (continued)

Pollutant	NO_2 Concentration µg/m³	NO_2 Concentration ppm	Exposure	Species	Effect	Reference
NO_2	18,800 28,200	10 15	2 hr, *K. pneumoniae* challenge	Squirrel monkey, male and female	Decreased tidal volume, increased respiratory rate 2 to 4 hr post exposure. No enhancement by *K. pneumoniae*.	Henry et al., 1969
	65,800	35			No mortality. Less drastic effects on respiratory function.	
	94,000	50			Tidal and minute volume decreased by 4 hr. Mortality: 2/3 within 5 to 72 hr. Increased respiratory rate.	
	94,000	50	2 hr, chal with *K. pneumoniae* after 24 hr		Tidal volume decreased. Respiratory rate increased. Death within 72 hr.	
	94,000	50	2 hr		Respiratory rate increased 2-fold. Decreased tidal volumes. Minute volumes constant. Respiratory rates high for 72 hr, return to normal by 7 days.	
NO_2	19,200 to 21,400	10.2 to 11.4	Continuous, 12 mo	Cat	Increased total airway resistance and upstream resistance. Decrease in static lung compliance. Internal surface area unchanged.	Kleinerman et al., 1976
fly ash	10,000 to 10,200				No effect due to fly ash.	
elutriated dust of fly ash	2,100 to 1,600					
NO_2	28,200	15	Continuous, lifetime	Rat	Increased tidal volumes 50 to 350%. Minor increased resistance and decreased compliance 15 to 20 wk. Increased emphysema.	Freeman et al., 1972

TABLE 14-10. (continued)

Pollutant	NO_2 Concentration $\mu g/m^3$	ppm	Exposure	Species	Effect	Reference
NO_2	38,400	20.4	20 to 22 hr/day, 7 days/wk, 12 to 14 mo	Hamster	Increased total pulmonary resistance during passive ventilation and maximal airflow with concurrent decreased flow values. Return to normal within 3 mo. Static lung compliance unchanged. Decreased surface area.	Kleinerman, 1977
NO_2	56,000	30	15 min	Rabbit	Redistribution of lung perfusion resulting in reduced storage activity in peripheral zones of lung.	von Nieding et al., 1973
NO_2	56,400 to 65,800	30 to 35	7 to 10 days, followed by papain	Hamster	NO_2 + papain increased lung volumes. NO_2 + papain increased pulmonary resistance ($p < 0.05$). Pulmonary resistance unchanged by papain.	Niewoehner and Kleinerman, 1973

Henry et al. (1970) exposed male squirrel monkeys continuously to 18,800 and 9,400 μg/m^3 (10 and 5 ppm) NO_2 for 1 and 2 months, respectively. Elevation in minute respiratory volume due to increased tidal volume and respiratory rate was apparent by 2 weeks and persisted throughout exposure to 18,800 μg/m^3 (10 ppm) NO_2. At the end of the exposure period, monkeys were challenged with K. pneumoniae; 3 days later minute volume was markedly reduced. Only minor changes in minute respiratory volumes were noted in monkeys exposed to 9,400 μg/m^3 (5 ppm) NO_2 for 2 months. The tidal volumes displayed a gradual reduction during the 2 months, while at the same time respiratory rates increased. After challenge with bacteria, minute volumes decreased and remained depressed.

Fenters et al. (1973) found that monkeys exposed to 1,880 μg/m^3 (1 ppm) NO_2 for 16.5 months showed little change in tidal volume, minute volume, and respiration rate. Subsequent challenge with influenza virus A/PR/8/34 produced no subsequent alterations either.

Environmental factors, such as heat stress, in combination with NO_2 exposure have been examined. (Coate and Badger, 1974) Monkeys continuously exposed to 9,400 and 18,800 μg/m^3 (5 and 10 ppm) NO_2 for 90 days were stressed at a temperature of 31°C versus 24°C for controls. At the higher concentration, NO_2 impaired the distribution of ventilation of the lungs, increased respiratory rates, and decreased tidal volumes. The addition of heat stress did not further impair distribution of ventilation, but it did decrease dynamic compliance of the lungs; NO_2 alone did not. No synergistic effect was seen at NO_2 concentrations of 9,400 μg/m^3 (5 ppm) with heat.

Freeman and Juhos (1976) exposed pregnant monkeys to NO_2 continuously and raised their offspring in similar environments. Adult and juvenile monkeys were exposed to 3,800 and 16,900 μg/m^3 (2 and 9 ppm) NO_2 for 10 and 5 years, respectively. No changes due to exposure could be seen in mean respiratory rate or functional residual capacity in the juveniles.

14.2.3.5 Studies of Hyperplasia--Chronic NO_2 exposure produces a transient hyperplasia of the Type II cells of the lung. This hyperplasia has stimulated inquiries into the potential for neoplasia or tumor formation due to NO_2. (See Table 14-11.)

The studies by Ide and Otsu (1973) revealed evidence of some tumor production in mice exposed to 9,400 to 18,800 μg/m^3 (5 to 10 ppm) NO_2 for 2 hours/day, 5 days/week for 50 weeks after receiving injections of 0.25 mg 4-nitroquinoline-1-oxide (a lung-tumor-specific carcinogen), but NO_2 did not enhance the tumor production (i.e., NO_2 had no synergistic or inhibitory properties with a known carcinogen). No tumors were observed in mice exposed to NO_2 alone. These data are of questionable value for predicting potential interactions with the broader classes of carcinogens.

Rejthar and Rejthar (1974) exposed rats to 9,400 μg/m^3 (5 ppm) NO_2 continuously for periods of 3, 5, 7, 9, and 11 weeks. The rats were then killed. Following a 3-week exposure, the bronchioles contained uniform cuboidal one-layer epithelium composed of nonciliated cells. The cells showed vacuolization, and hyperplastic foci appeared in the bronchiolar epithelium. The foci were 2- to 4-layer pyramidal formations. By 5 weeks, extensive hyperplasia composed of three to four layers of epithelial cells was apparent. Centers of cuboidal metaplasia were

TABLE 14-11. STUDIES OF POTENTIAL HYPERPLASIA

Pollutant	Pollutant Concentration μg/m³	ppm	Exposure	Species	Effect	Reference
NO_2 + CO	940 to 1,500 + 58,000	0.5 to 0.8 + 50	Continuous, 30 days	Mouse	Increased hyperplasia terminal bronchioles to alveolus. No difference from NO_2 alone. CO (115,000 μg/m³; 100 ppm) alone for 30 days failed to induce hyperplasia.	Nakajima et al., 1972
Synthetic Smog NO_2 CO O_3 SO_2	 1,500 5,750 760 5,700	 0.8 5 0.38 2.2	23 to 24 hr/day, 8 to 12 mo	Mouse	By 20 days exposure, increased thickened bronchial membranes. By 60 days, very thick membranes appear to have villus-like hyperplastic folds. 4 months post-exposure, hyperplasia regressed towards normal.	Loosli et al., 1972
NO_2	2,360	1.26	12 hr/day, 3 mo	Rat, prior to breeding	No effect on fertility. Decrease in litter size and neonatal weight. No teratogenic effects.	Shalamberidze and Tsereteli, 1971
NO_2	9,400	5	Continuous, to 11 wk	Rat	Appearance of hyperplastic foci in the shape of 2 to 4 layer pyramids by 3 wk. Decreased ciliated cells. Extensive hyperplasia (3 to 4 layers of epithelium), cuboidal metaplasia in adjacent alveoli by 5 wk. Hyperplasia in all bronchioles, decreased bronchiolar lumina, polymorphous epithelium extensive by 7 wk. Terminal bronchiolar epithelium contained only 2 or 3 irregular layers, increased number of ciliated cells by 9 wk. By 11 wk return to 1 layer epithelium. Lungs at indefinite state of repair from week 7 on.	Rejthar and Rejthar, 1974

TABLE 14-11. STUDIES OF POTENTIAL HYPERPLASIA

Pollutant	Pollutant Concentration μg/m^3	Pollutant Concentration ppm	Exposure	Species	Effect	Reference
NO_2 CO	940 to 1,500 + 58,000	0.5 to 0.8 + 50	Continuous, 30 days	Mouse	Increased hyperplasia terminal bronchioles to alveolus. No difference from NO_2 alone. CO (115,000 μg/m^3; 100 ppm) alone for 30 days failed to induce hyperplasia.	Nakajima et al., 1972
Synthetic Smog NO_2 CO O_3 SO_2	 1,500 5,750 760 5,700	 0.8 5 0.38 2.2	23 to 24 hr/day, 8 to 12 mo	Mouse	By 20 days exposure, increased thickened bronchial membranes. By 60 days, very thick membranes appear to have villus-like hyperplastic folds. 4 months post-exposure, hyperplasia regressed towards normal.	Loosli et al., 1972
NO_2	2,360	1.26	12 hr/day, 3 mo	Rat, prior to breeding	No effect on fertility. Decrease in litter size and neonatal weight. No teratogenic effects.	Shalamberidze and Tsereteli, 1971
NO_2	9,400	5	Continuous, to 11 wk	Rat	Appearance of hyperplastic foci in the shape of 2 to 4 layer pyramids by 3 wk. Decreased ciliated cells. Extensive hyperplasia (3 to 4 layers of epithelium), cuboidal metaplasia in adjacent alveoli by 5 wk. Hyperplasia in all bronchioles, decreased bronchiolar lumina, polymorphous epithelium extensive by 7 wk. Terminal bronchiolar epithelium contained only 2 or 3 irregular layers, increased number of ciliated cells by 9 wk. By 11 wk return to 1 layer epithelium. Lungs at indefinite state of repair from week 7 on.	Rejthar and Rejthar, 1974

found in adjacent alveoli. By 7 weeks, hyperplasia was apparent in all bronchioles, thus narrowing the bronchiolar lumina. Polymorphous epithelium was extensive with a few ciliated cells in hyperplastic areas. After 9 weeks, terminal bronchiolar epithelium generally showed two or three irregular layers. The number of ciliated cells increased, but cilia were often located atypically in intercellular spaces. A return to a single layer of epithelium without cilia was observed after 11 weeks. Seven weeks after exposure to NO_2, the lungs appeared to be in a state of repair moving towards reversal of the lesions.

Nakajima et al. (1972) exposed mice to 940 to 1,500 $\mu g/m^3$ (0.5 to 0.8 ppm) NO_2 for 30 days. Examination revealed hyperplasia from the terminal bronchiole to the alveolus. Mice exposed to the same concentrations of NO_2 with CO (58,000 $\mu g/m^3$; 50 ppm) for 30 days revealed the same hyperplastic foci in the terminal bronchioles. At exposure concentrations up to 115,000 $\mu g/m^3$ (100 ppm) for 30 days, CO by itself failed to induce hyperplasia in mice.

Stupfel et al. (1973) exposed rats to automotive exhaust 6 hours/day, 5 days/week for periods of 2.5 months to 2 years. The exhaust contained 58,000 $\mu g/m^3$ (50 ppm) CO, two different concentrations of NO_x (0.2 and 23 ppm), CO_2 (0.07 and 0.37 percent), along with aldehydes (0.1 and 2 ppm). No effects were observed at 0.2 ppm NO_x. At 23 ppm NO_x, more spontaneous tumors and cutaneous abscesses as well as bilateral renal sclerosis were seen.

14.2.3.6 Edemagenesis and Tolerance--Sherwin and Carlson (1973) reported an increase of protein in the lavage fluid from lungs of guinea pigs with an unquantified vitamin C deficiency exposed continuously to 750 $\mu g/m^3$ (0.4 ppm) NO_2 for 1 week ($p < 0.001$). (See Table 14-12.) Proteins were identified and measured by disc electrophoresis. No remarkable differences were noted in the composition of the filtered proteins obtained by pulmonary lavage. Mice injected with ^3H-rabbit albumin accumulated ^3H in their lungs following 14 day, continuous exposure to 7,500 to 13,000 $\mu g/m^3$ (4 to 7 ppm) NO_2. (Sherwin and Richters, 1971) Using injected horseradish peroxidase as a marker, this group of researchers recently reported increased retention of protein in pulmonary air spaces after exposure to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 5 days/week for 3 weeks (Sherwin et al., 1977). Greater retention of horseradish peroxidase occurred after 6 weeks of exposure.

Selgrade et al. (1981) reexamined this problem focusing on the role of vitamin C deficiency in promoting of NO_2-induced edema. (See "Section 14.2.3.2.2 Lipid and Diet Effects" for a detailed description of the concentrations, times and effects.) They found that vitamin C depletion enhanced both edema formation, as measured by protein and lipid content of the lavage fluid, and increased mortality at 9400 $\mu g/m^3$ (5.0 ppm). There are differences in the two studies, however. Selgrade et al. found no effect of exposure to 752 $\mu g/m^3$ (0.4 ppm) for up to 1 week whereas Sherwin and Carlson (1973) did. Selgrade et al. also found a new protein in the lavage fluid not found in serum and found that the lipid content, while increased, did not reflect that of the serum. Sherwin and Carlson used disc gel electrophoresis to measure protein content while Selgrade et al. used the microchemical Lowry method. The qualitative difference in identification of proteins may also have resulted from the use of gel scanning by Selgrade et al. and the improvements in gel resolution brought

TABLE 14-12. PRODUCTION OF LUNG EDEMA BY NO_2

Pollutant Concentration		Duration	Species	Effects	Reference
$\mu g/m^3$	ppm				
750	0.4	Continuous, 1 wk	Guinea pig	Increased proteins in lung lavage fluid of exposed animals with an unquantified vitamin C deficiency detected by disc gel electrophoresis.	Sherwin and Carlson, 1973
940	0.5	5 days/wk, 3 or 6 wk	Mouse	Horseradish peroxidase used as a marker for proteins showed greater sequestering in exposed mice at 3 wk than 6 wk. Suggests edema.	Sherwin et al., 1977
7,500 to 13,000	4 to 7	Continuous, 14 days	Mouse	^3H rabbit albumin infiltration indicates lung edema.	Sherwin and Richters, 1971
56,400	30	Continuous, to 30 days	Hamster	Lung wet weight elevated at 1 and 30 days.	Kleinerman and Rynbrandt, 1976

about in recent developments in this field. The major difference may lie, however, in the degree of vitamin C depletion of the guinea pigs in the two studies. Those of Sherwin and Carlson are not documented with blood vitamin C levels. Selgrade et al. found only mild depletion was necessary. Consequently, future studies will be needed to clarify this point. Taken together, these two studies and those reported above suggest that NO_2 damage to the lung can be modified by the dietary intake and that lavage fluid composition is a sensitive measure of damage to the lung.

The development of tolerance to lethal concentrations of NO_2 has been correlated with lethal edema production. Wagner et al. (1965) examined the question of tolerance along with several other characteristics. They found that tolerance could be evoked by prior exposure to low or high concentrations of NO_2, in young and old animals. (See Table 14-13) Mice were made tolerant to an LC50 dose of 113,000 μg/m^3 (60 ppm) by prior exposure to 9,400 μg/m^3 (5 ppm) NO_2 for 7 weeks (hours/day not specified). Tolerance disappeared after 3 months following removal from the NO_2 exposure. Rats also were made tolerant.

14.2.4. Extrapulmonary Effects

14.2.4.1 Nitrogen Dioxide-induced Changes in Hematology and Blood Chemistry--Exposure of experimental animals and humans to NO_2 alone or in combination with other pollutants produces an array of hematological perturbations (see Table 14-14) with a biological significance which is not easily interpretable (see Chapter 15 for human studies).

Shalamberidze (1969) exposed rats continuously to 100 μg/m^3 (0.05 ppm) NO_2 for 90 days with no change in blood hemoglobin or erythrocyte levels.

A 7-day exposure to 940 μg/m^3 (0.5 ppm) NO_2 resulted in a depression in GSH peroxidase levels of RBC in guinea pigs ($p < 0.001$) which was not observed after exposure for 4 months (Donovan et al., 1976; Menzel et al., 1977).

Kosmider et al. (1975) exposed guinea pigs for 8 hours/day for 120 days to 940 μg/m^3 (0.5 ppm) NO_2 with 1,000 μg/m^3 (0.39 ppm) SO_2 or 940 μg/m^3 (0.5 ppm) NO_2 with 1,000 μg/m^3 (0.39 ppm) SO_2 and 70 μg/m^3 (0.1 ppm) ammonia. Animals exposed to NO_2 and SO_2, with and without ammonia, displayed an increase in white blood cells (WBC) and a decrease in RBC and hemoglobin. Following exposure, a differential count of white cells revealed a decrease in neutrophils and eosinophils and an increase in lymphocytes.

Mersch et al. (1973) exposed guinea pigs to 680 μg/m^3 (0.36 ppm) NO_2 continuously for 1 week. Following exposure, RBC D-2,3-diphosphoglycerate was significantly increased ($p < 0.05$), a measure which could reflect tissue deoxygenation.

Studies reported by Nakajima and Kusumoto (1970) showed that addition of 58,000 μg/m^3 (50 ppm) CO to 940 to 1,500 μg/m^3 (0.5 to 0.8 ppm) NO_2 did not change the carboxyhemoglobin concentration in the blood of mice exposed 24 hours/day for 1 to 1.5 months to CO alone. They also exposed mice to 1,500 μg/m^3 (0.8 ppm) NO_2 for 5 days and found that methemoglobin levels were not affected (Nakajima and Kusumoto, 1968).

Mitina (1962) exposed rabbits to 2,400 to 5,640 μg/m^3 (1.3 to 3 ppm) NO_2 and/or 5,240 μg/m^3 (2 ppm) SO_2 2 hours/day for 15 and 17 weeks. Exposure to NO_2 alone produced a significant rise

TABLE 14-13. TOLERANCE TO NO_2 EXPOSURES

NO_2 Concentration μg/m^3	ppm	Duration	Species	Effects	Reference
9,400	5	7 wk	Mouse	Challenge with LC50 dose (113,000 μg/m^3 (60 ppm) NO_2 for 5 hr, 24 hr post-exposure) caused 28% less mortality than in naive mice.	Wagner et al., 1965
9,400 + 47,000	5 + 25	13 mo + 6 wk	Rat	Challenge with NO_2 (132,000 μg/m^3 (70 ppm) for 5 hr, 3 days post-exposure) caused 0% mortality compared to 67% in pre-exposed controls.	
47,000	25	7 wk	Mouse	Challenge with NO_2 (132,000 μg/m^3 (70 ppm) for 5 hr, 3 days post-exposure) caused no mortality 24 hr post-challenge compared to 29% in naive mice. Tolerance disappeared in 1 mo.	
18,800	10	5 hr/day, 5 days/wk,	Hamster	Tolerance developed to normally lethal 5 hr 18,800 μg/m^3 (10 ppm) NO_2 exposure. Protection against further cytological injury but not against the cytotoxic effects of >18,800 μg/m^3 (10 ppm) as measured by increased DNA synthesis.	Creasia, 1978
		5 hr/day, 1 day/wk, 3 wk		Tolerance to normally lethal 131,600 μg/m^3 (70 ppm) NO_2. Protection against further cytological injury.	
47,000	25	6 hr/day, 2 days	Rat	Increased tolerance to 141,050 μg/m^3 (75 ppm) NO_2. Increased G-6-PD, catalase, 41% increase cytochrome oxidase. No effect on superoxide dismutase.	Crapo et al., 1978

TABLE 14-14. NITROGEN DIOXIDE-INDUCED CHANGES IN HEMATOLOGY

	Pollutant Concentration					
Pollutant	$\mu g/m^3$	ppm	Exposure	Species	Effects	Reference
NO_2	100	0.05	Continuous, 90 days	Rat	No effect on blood hemoglobin or erythrocytes.	Shalamberidze, 1969
NO_2	680	0.36	Continuous, 7 days	Guinea pig	Increased red blood cell D-2,3-diphosphoglycerate.	Mersch et al., 1973
NO_2	940	0.5	8 hr/day, 7 days	Guinea pig	Decrease in RBC GSH peroxidase ($p < 0.001$).	Donovan et al., 1976
			8 hr/day, 4 mo		No change in RBC GSH peroxidase.	Menzel et al., 1977
NO_2 SO_2	940 + 1,000	0.5 + 0.39	8 hr/day, 120 days	Guinea pig	Same effects in both exposures. Increased WBC and lymphocytes. Decreased RBC, hemoglobin, neutrophils and eosinophils.	Kosmider et al., 1975
NO_2 SO_2 NH_3	940 + 1,021 + 70	0.5 + 0.39 + 0.1				
NO_2 CO	940 to 1,500 + 58,000	0.5 to 0.8 + 50	Continuous, 1 to 1.5 mo	Mouse	Addition of CO to NO_2 failed to affect carboxyhemoglobin.	Nakajima and Kusumoto, 1970
NO_2	1,500	0.8	Continuous, 5 days	Mouse	No effect on methemoglobin.	Nakajima and Kusumoto 1968
NO_2	1,880	1	Continuous, 493 days followed by influenza A/PR/8/34 virus	Squirrel monkey	No effect on hematocrit, hemoglobin, or clinical biochemical parameters. Following viral challenge increased leukocyte count.	Fenters et al., 1973

TABLE 14-14. (continued)

Pollutant	Pollutant Concentration µg/m^3	 ppm	Exposure	Species	Effects	Reference
NO NO_2 NO NO_2	940 1,880 1,840 + 2,450	0.8 1 1.5 + 1.3	16 hr/day, 7 days/wk, 4 yr	Dog	No effects on hematocrit viscosity, carboxyhemoglobin, or methemoglobin.	Block et al., 1973
NO_2	2,400 to 5,640	1.3 to 3	2 hr/day, 15 & 17 wk	Rabbit	Increased leukocytes followed by decreased phagocytic activity. Decreased RBC.	Mitina, 1962
SO_2	5,240	1.9	2 hr/day, 15 wk		Decreased phagocytic activity leukocyte. No effect on RBC.	
NO_2 SO_2	2,400 + 5,000	1.3 + 1.9	2 hr/day, 15 wk		No effect on RBC.	
NO_2 NaCl	3,760 + 330	2 + 0.14	Continuous, 14 mo	Monkey, rat	Monkey: with or without NaCl, hypertrophy of respiratory bronchiolar epithelium. Rat: with or without NaCl, polycythemia with reduced mean corpuscular volume and normal mean corpuscular hemoglobin concentration. Neutrophil/lymphocyte ratio tendency to shift upwards in both animal species tested.	Furiosi et al., 1973
NO_2	9,400 18,800	5 10	Continuous, 90 days	Cynomolgus monkey, male	No effect on hematological parameters.	Coate and Badger, 1974
NO_2	45,100	24	4 hr	Rabbit	Increased nitrite & nitrate in blood. Thought to react with hemoglobin producing methemoglobin.	Svorcova and Kaut, 1971
NO_2	48,900	26	191 days	Dog	Increased WBC disappeared following cessation of NO_2. Decreased hematocrit and hemoglobin; increased mean corpuscular volume and mean corpuscular hemoglobin.	Lewis et al., 1973
NO_2	73,000 to 310,000	39 to 164	5 to 60 min	Dog	No effect on hematocrit or platelet counts.	Carson et al., 1962

in leukocytes followed by a decrease in their phagocytic activity. Exposure to NO_2 alone also reduced the number of RBC, while a mixture of NO_2 and SO_2 or SO_2 alone had no effect.

Fenters et al. (1973) showed that exposing male squirrel monkeys to 1,880 $\mu g/m^3$ (1 ppm) NO_2 continuously for 493 days had no significant effect on hematocrit, hemoglobin, total protein, globulins, chloride, sodium, calcium, potassium, glucose, blood urea, nitrogen, glutamic-pyruvic transaminase, lactate dehydrogenase, and lactate dehydrogenase isoenzymes. Challenge with influenza A/PR/8/34 increased leukocytes in NO_2-exposed animals above levels in similarly challenged controls.

Coate and Badger (1974) exposed monkeys to 9,400 and 18,800 $\mu g/m^3$ (5 and 10 ppm) NO_2 for 90 days with no direct effect on hematological parameters. In another study, monkeys and rats were exposed to 330 $\mu g/m^3$ (0.14 ppm) NaCl and 3,760 $\mu g/m^3$ (2 ppm) NO_2 for 14 months (Furiosi et al., 1973). Exposure to NO_2 with or without NaCl produced polycythemia with reduced mean corpuscular volume and approximately normal mean corpuscular hemoglobin concentration. In monkeys and rats exposed to NO_2 with and without NaCl, the ratio of neutrophils to lymphocytes was greater.

Block et al. (1973) conducted several hematological studies on dogs exposed 16 hours/day, 7 days/week, for 4 years to 940 $\mu g/m^3$ (0.76 ppm) NO, 1,880 $\mu g/m^3$ (1 ppm) NO_2, or 1,840 $\mu g/m^3$ (1.5 ppm) NO plus 2,450 $\mu g/m^3$ (1.3 ppm) NO_2. No changes in hematocrit, viscosity, carboxyhemoglobin, or methemoglobin were found.

14.2.4.2 Central Nervous System and Behavioral Effects--Information regarding the effects of NO_2 on the central nervous system or on animal behavior is limited to a few studies, (see Table 14-15), most of which have uncertain relationships to humans.

Tusl et al. (1973) exposed rats to 9,400 $\mu g/m^3$ (5 ppm) NO_2 for 8 weeks. The influence of NO_2 on swimming of rats was measured. By the 5th and 6th weeks of exposure, swimming performance had decreased 25 percent. In rats exposed to 1,880 $\mu g/m^3$ (1 ppm) NO_2, performance was maintained with a slight tendency toward deterioration.

Yakimchuk and Chelikanov (1968) reported that rats exposed to 600 $\mu g/m^3$ (0.32 ppm) NO_2 for 3 months developed a delay in their conditioned reflexes to sound and light. Shalamberidze (1969) exposed rats to 100 $\mu g/m^3$ (0.05 ppm) NO_2 for 3 months with no demonstrated effects on the central nervous system.

Exposure of guinea pigs to 1,000 $\mu g/m^3$ (0.53 ppm) NO_2 8 hours/day for 180 days affected brain enzyme levels (Drozdz et al., 1975). Decreases were seen in brain malate dehydrogenase, alanine aminotransferase, sorbitol dehydrogenase, lactate dehydrogenase, adenosine triphosphatase, and 5'-nucleotidase. Increases were seen in 1,6-diphosphofructose aldolase, isocitrate dehydrogenase, a-hydroxybutyrate dehydrogenase, phosphocreatine kinase, and cholinesterase.

14.2.4.3 Biochemical Markers of Organ Effects--A major goal has been the detection of early enzymatic markers of NO_2 effects. Studies of enzyme levels in different animal species indicate that the earliest enzymatic changes of nitrogen dioxide effects occur in guinea pigs. (See Table 14-16) Several such marker enzymes have been determined in blood. Release of

TABLE 14-15. CENTRAL NERVOUS SYSTEM AND BEHAVIORAL EFFECTS

Pollutant	Pollutant Concentration µg/m³	Pollutant Concentration ppm	Exposure	Species	Effect	Reference
NO_2	100	0.05	Continuous, 3 mo	Rat	No effect on CNS.	Shalamberidze, 1969
NO_2	1,000	0.53	8 hr/day, 180 days	Guinea pig	Decreased malate, sorbitol, lactate dehydrogenase; alanine aminotransferase; AlPase and 5'-nucleotidase. Increased 1,6-diphosphofructose aldolase; isocitrate, and alpha-hydroxybutyrate dehydrogenase; phosphocreatine kinase and cholinesterase.	Drozdz et al., 1975
NO_2	600	0.32	Continuous, 3 mo	Rat	Decreased conditioned reflexes to sound and light.	Yakimchuk and Chelikanov, 1968
NO_2	1,880	1	20 min/day, to 6 mo	Rat	More or less constant swimming performance only slight tendency to deterioration.	Tusl et al., 1973
	9,400	5			Swimming performance decreased 25% by 5th and 6th wk of exposure	
	37,600	20			Swimming velocity declined from 1st mo.	
	6,580	3.5	6 hr/day, 8 wk		Decreased swimming performance.	
NO_2	14,000	7.7	6 hr	Mouse, male	Decreased voluntary running activity. Return to normal within 24 hr post exposure.	Murphy et al., 1964
NO_2	75,000 37,600 15,040	40 20 8	5 hr 1 day 19 days	Rat	20% decrease swimming endurance. 10% decrease swimming endurance. 5% decrease swimming endurance.	Campbell, 1976
Auto Exhaust CO NO_x CO_2 (0.07 and 0.37% Aldehydes	58,000	50 (0.2 and 23) (0.1 and 2.0)	6 hr/day, 5 days/wk, 2.5 mo to 2 yr	Rat, male	Decreased sound avoidance reflexes, learning rate lowered.	Stupfel, 1973

TABLE 14-16. BIOCHEMICAL MARKERS OF ORGAN EFFECTS

NO_2 Concentration µg/m³	NO_2 Concentration ppm	Exposure	Species	Effect	Reference
470 to 9,400	0.25 to 5	3 hr	Mouse	Increase in pentobarbital-induced sleep time in female mice only. Repeated daily exposures caused no effect.	Miller et al., 1980
940	0.5	8 hr/day, 7 days	Guinea pig	Serum lactic dehydrogenase, total creatinine phosphokinase, SGOT, and SGPT, cholinesterase and lysozyme elevated.	Menzel et al., 1977
		8 hr/day, 4 mo.		Lysozyme and cholinesterase depressed.	
940	0.5	Continuous, 7-14 days	Guinea pig	Albumin and globulins found in urine.	Sherwin and Layfield, 1974
1,000	(NO_x; mainly NO_2)	8 hr/day, 180 days	Guinea pig	Nitrates and nitrites excreted in urine. Serum cholesterol slightly elevated; total lipids depressed. Urinary Mg increased while liver and brain Mg decreased. Hepatic edema reported.	Kosmider, 1975
1,880	1	Continuous, 6 mo	Guinea pig	Protein synthesis inhibited. Body weight, total serum proteins, and immunoglobulins decreased.	Kosmider et al., 1973a
2,000	1.05	8 hr/day, 180 days	Guinea pig	Plasma changes: Decreased albumin, seromucoid, cholinesterase, alanin, and aspartate transminases. Increased $alpha_1$ and $beta_2$ immunoglobulins. Intracellular edema of liver. Hepatic changes similar to plasma.	Drozdz et al., 1976

TABLE 14-16. (continued)

NO_2 Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
11,660	6.2	Continuous, 4 days	Rat	No effect on serum lysozyme.	Chow et al., 1974
47,000 to 179,000	25 to 95	2 hr	Rat	Plasma corticosterone increased proportional to No_2 concentration from 47 to 179 mg/m³; at 85 mg/m³ x 5 hr/day levels returned to normal in 19 days; at 56 mg/m³ x 5 hr/day levels returned to normal in 5 days.	Tusl et al., 1975
56,400	30	Continuous, 30 days	Hamster	Serum antiprotease levels increased	Kleinerman and Rynbrandt, 1976 Rynbrandt and Kleinerman, 1977

lysozyme into the blood from pulmonary damage was cited above (Chow et al., 1974; Donovan et al, 1976; Menzel et al., 1977). The CPK isoenzyme patterns seen in NO_2-exposed guinea pigs are difficult to differentiate from CPK patterns caused by myocardial damage and are not particularly useful (Donovan et al., 1976). Plasma cholinesterase (CHE) was significantly ($p < 0.001$) elevated after a 7-day exposure to 940 $\mu g/m^3$ (0.5 ppm) NO_2 but fell on long-term exposure (4 months) ($p < 0.001$). CHE levels are indicators of hepatic and myocardial disease, being elevated in hemochromatosis, and usually depressed during active hepatocellular disease (MacQueen and Plant, 1973; Moore et al., 1957). Increased CHE is seen with cardiac surgery, but values are depressed consistently with myocardial infarction (MacQueen and Plant, 1973). Aside from metastatic carcinoma from the liver to the lung, pulmonary disease including pneumonia has not been previously associated with changes in CHE levels. Most likely, the alterations in SGOT, SGPT, and LDH reported in the guinea pig studies are related to NO_2-induced hepatic damage. (Donovan et al., 1976) The persistent alteration in CHE, albeit lower activity, after 4 months exposure to 940 $\mu g/m^3$ (0.5 ppm) NO_2 suggests an hepatic lesion.

Another study indicating hepatic damage (Drozdz et al., 1976) revealed decreased plasma levels of albumin, seromucoid, alanine and aspartate transaminases when guinea pigs were exposed to 2,000 $\mu g/m^3$ (1.05 ppm) NO_2 for 8 hours/day for 180 days. In agreement with Donovan et al., (1976) Drozdz et al. (1976) reported decreased plasma cholinesterase levels. This group also found increased serum levels of α_1- and β_2-immunoglobins. Electron micrographs of the liver suggested intracellular edema. Kosmider (1975) reported decreased serum cholesterol, total lipids, and beta and gamma lipoproteins in guinea pigs exposed to 1,000 $\mu g/m^3$ NO_x (mainly NO_2), 8 hours/day for 120 days. At the same time, there was an increase in serum alpha (α) lipoproteins. Blood serum sodium and magnesium were reduced while liver and brain were depleted of magnesium and zinc as well. Cell permeabilities were changed and ions displaced. Edema of the liver mitochondria occurred. At the same time, urine had increased secretions of nitrite, nitrate, and coproporphyrin. Respiratory rate rose significantly with little or no excitement or aggressiveness. Kosmider also exposed these animals to the same schedule of NO_x and added ammonia. Nitrogen oxides reacted with ammonia (1,000 $\mu g/m^3$; 1.4 ppm), reducing the lipid and electrolyte disturbances seen with NO_x exposure alone. Blood serum lipids, lipoproteins, and cholesterol were not significantly altered from those of control guinea pigs breathing filtered clean air. There was a decrease in the urinary excretion of nitrites, nitrates, and coproporphyrin from NO_x-ammonia treated animals. Blood serum sodium and potassium were lowered, while magnesium and calcium were higher than controls. No effect on serum calcium levels was seen with NO_x alone. Liver mitochondria contracted, in contrast to the edematous state seen in NO_x-exposed groups. Earlier, Kosmider et al. (1973a) also reported a general decrease in protein synthesis evidenced by decreased serum proteins and declining body weights of guinea pigs exposed continuously to 1,880 $\mu g/m^3$ (1 ppm) NO_2 for 6 months.

An interesting observation of proteinuria in the guinea pig was reported by Sherwin and Layfield (1974) who found consistently higher levels of urinary protein ($p < 0.01$) in animals breathing 940 $\mu g/m^3$ (0.5 ppm) NO_2 continuously for 7 to 14 days. Proteinuria was detected in another group of animals exposed for 4 hours per day to 750 $\mu g/m^3$ (0.4 ppm) NO_2 ($p < 0.05$). Disc electrophoresis of the urinary proteins demonstrated the presence of albumin, and alpha, beta, and gamma globulins. The presence of high molecular weight proteins in urine is characteristic of the nephrotic syndrome. Histopathological studies of the kidney were negative.

The influence of NO_2 on pentobarbital (PEN)-induced sleeping time was investigated in mice. (Miller et al., 1980) A 3-hour exposure to concentrations as low as 470 $\mu g/m^3$ (0.25 ppm) NO_2 caused a significant increase in PEN-induced sleeping time in female mice. No significant effects on PEN-induced sleeping time in females were detected after 1 or 2 days (3 hr/day) exposure to 235 $\mu g/m^3$ (0.125 ppm) NO_2. None of the exposure regimens affected the time required for the drug to induce sleep. Two or three days (3 hr/day) exposure to 470 $\mu g/m^3$ (0.25 ppm) NO_2 caused no significant effect in female mice. This trend of a reduction or disappearance of the effect after repeated exposure was observed at other, higher, concentrations also. When the effects of repeated daily exposures (3 hr/day) to 9,400 $\mu g/m^3$ (5 ppm) NO_2 were compared in male and female mice, the females had a significantly increased PEN-induced sleeping time after 1 and 2 days of exposure. However, a significant increase in PEN-induced sleeping time was not observed in males until the 3rd day of exposure. Since the duration of PEN-induced sleeping time is primarily determined by hepatic mixed function oxidase activity, it is possible that NO_2 may alter some aspects of xenobiotic metabolism.

Ozone (Graham, 1979) also increased PEN-induced sleeping time in female, but not male, mice. Unlike NO_2 however, as the concentration was decreased, an increased number of daily 3-hr exposures were needed to observe the effect. For example, at 490 $\mu g/m^3$ (0.25 ppm) O_3, a significant effect was observed only after 6 and 7 days (3 hr/day) of exposure. Thus, for this model system, a single exposure of NO_2 caused more effect than a single exposure to O_3.

14.2.4.4 Teratogenesis and Mutagenesis--There is little or no evidence in the literature demonstrating that exposure to NO_2 is teratogenic or mutagenic in animals. Shalamberidze and Tsereteli (1971) exposed rats to 2,360 $\mu g/m^3$ (1.3 ppm) NO_2, 12 hours/day for 3 months at which time exposure ceased and the animals were bred. Long-term NO_2 exposure had no effect on fertility. There was a statistically significant decrease in litter size and neonate weight ($p < 0.001$). *In utero* death due to NO_2 exposure resulted in smaller litter sizes, but no direct teratogenic effects were observed in the offspring. In fact, after several weeks, NO_2-exposed litters approached weights similar to controls. (See Table 14-17.)

Gooch et al. (1977) exposed C3H male mice to 190, 1,880, 9,400, and 18,800 $\mu g/m^3$ (0.1, 1, 5, and 10 ppm) NO_2 for 6 hours. Blood samples were obtained at 0 time, and 1 week and 2 weeks post-exposure. Mouse leukocyte chromosomal analysis revealed that NO_2 did not increase chromatid- or chromosome-type alterations. The analysis of primary spermatocytes showed no direct effect of NO_2 exposure on their chromosomes. Therefore, in these experiments, NO_2 exposure did not induce mutagenesis. (See Table 14-17.)

TABLE 14-17. STUDIES OF POTENTIAL MUTAGENESIS, TERATOGENESIS

Pollutant	Pollutant Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
NO_2	190 1,880 9,400 18,800	0.1 1 5 10	6 hr	Mouse	2 wk post-exposure no increase in chromatid or chromosome-type alterations in leukocytes or primary spermatocytes. No mutagenic effects noted.	Gooch et al., 1977
NO_2	9,400	5	Continuous, to 11 wk	Rat	Appearance of hyperplastic foci in the shape of 2 to 4 layer pyramids by 3 wk. Decreased ciliated cells. Extensive hyperplasia (3 to 4 layers of epithelium), cuboidal metaplasia in adjacent alveoli by 5 wk. Hyperplasia in all bronchioles, decreased bronchiolar lumina, polymorphous epithelium extensive by 7 wk. Terminal bronchiolar epithelium contained only 2 or 3 irregular layers, increased number of ciliated cells by 9 wk. By 11 wk return to 1 layer epithelium. Lungs at indefinite state of repair from week 7 on.	Rejthar and Rejthar, 1974
NO_2	9,400 to 18,800	5 to 10	2 hr/day, 5 day/wk, 50 wk	Mouse	Mice given 4-nitroquinoline-1-oxide and NO_2; NO_2 had no effect on tumor production.	Ide and Otsu, 1973
NO_2	18,800	10	2 hr/day, 5 day/wk, 50 wk	Mouse	Mice given 4-nitroquinoline-1-oxide (carcinogenic agent) + NO_2 decreased incidence of lung tumors.$_2$	Otsu and Ide, 1975
NO_2	18,800	10	Continuous from pregnancy to 3 mo after delivery	Rat	Decreased litter size and increased mortality of neonates up to 15 days post delivery. No teratogenic effects noted.	Freeman et al., 1974b

TABLE 14-17. (continued)

Pollutant	Pollutant Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
Auto Exhaust						
CO	58,000	50	6 hr/day, 5 days/wk, 2.5 mo to 2 yr	Rat	Auto exhaust had no biological effects when NO_x was 0.2 ppm. Exposure to NO_x (23 ppm) increased number of spontaneous tumors, cutaneous abscesses, and bilateral renal sclerosis. No tumors or abscesses in lungs.	Stupfel et al., 1973
NO_x		(0.2 and 23)				
CO_2 (0.07 and 0.37%)						
Aldehydes		(0.1 and 2.0)				

14.2.4.5 Effects of NO_2 on Body Weights--Dogs, rabbits, guinea pigs, rats, hamsters, and mice have been exposed to 100 to 47,000 $\mu g/m^3$ NO_2 (0.05 to 25 ppm) up to 18 months without reported body weight loss (Nakajima et al., 1972; Shalamberidze, 1969; Wagner et al., 1965). (See Table 14-18.) Oda et al., (1973) however, observed reduced body weights of rats exposed to ambient air containing 135 $\mu g/m^3$ (0.07 ppm) NO_2 amongst other pollutants over a 100-day period.

14.3 DIRECT EFFECT OF COMPLEX MIXTURES

The oxidation of organic substances contained in solid, liquid, and gaseous fuels, as well as the reaction of atmospheric oxygen and atmospheric nitrogen at furnace temperatures, are sources for complex mixtures of nitrogen oxides in the atmosphere. Vehicles and power plants release large amounts of nitrogen oxides into the atmosphere. The biological and/or toxicological effects of the combination of these nitrogen oxides with other pollutants is the scope of this section. Much of the research reported here was conducted with complex pollutant mixtures and had no single-pollutant controls. Thus, the precise contribution of the given level of NO_2 to the effects observed is not possible to discern. However, in some of these studies, NO_x concentration was varied between groups or NO_x was present in one group and absent in another. In these instances, the influence of NO_x can be elucidated.

Emik et al. (1971) noted that males of the C57Bl/6 strain of mice exposed to ambient air in California died sooner than either C57Bl/6 females or males and females of the A and A/J strains exposed to clean, filtered air. Guinea pigs and other strains of mice demonstrated no difference in survival when exposed to clean or polluted air. Concentrations of about 40 $\mu g/m^3$ (0.02 ppm) NO_2 were observed during the study. A number of other pollutants were also present.

Loosli et al. (1972) exposed specific pathogen-free mice to synthetic smog 23 to 24 hours/day for 8 to 12 months. The smog was composed of 1,500 $\mu g/m^3$ (0.8 ppm) NO_2, 5,750 $\mu g/m^3$ (5 ppm) CO, 760 $\mu g/m^3$ (0.38 ppm) O_3, and 5,700 $\mu g/m^3$ (2.2 ppm) SO_2. The lungs of mice exposed for 20 days had thickened bronchial membrane due to cell proliferation. By 60 days the membranes were markedly thickened and appeared to have villus-like hyperplastic folds, whereas evidence of hyperplasia in control animals was absent. When mice were removed from smog exposure after 4 months, the hyperplasia regressed.

Hysell et al. (1975) studied the toxicological effects on animals of automotive emissions, with or without a catalytic converter and with or without irradiation. Female lactating rats and their 2-week-old offspring were exposed for 7 days. Infant mortality was increased following exposure to auto emissions containing 9,400 $\mu g/m^3$ (5 ppm) NO_2 without catalytic conversion, whereas mortality was not affected in animals exposed to emissions modified by catalytic conversion. Adult male rats and hamsters were exposed to these regimens for 6 days. An increase in hemolysis-resistant RBC due to high ambient CO followed exposure to raw exhaust with no catalyst. Blood eosinophils in rats exposed to converted exhaust were not affected. Animals developed extensive pulmonary changes when exposed to irradiated, unconverted auto exhaust. Hamsters developed purulent bronchitis, bronchiolitis, and broncho-

TABLE 14-18. EXTRAPULMONARY EFFECTS OF NO_2: BODY WEIGHT

NO_2 Concentration		Exposure	Species	Effect	Reference
μg/m³	ppm				
100	0.05	90 days	Rat	No effect.	Shalamberidze, 1969
135	0.072	Continuous, 100 days	Rat	Ambient air exposure (6.3 ppm CO, 0.206 ppm NO, 0.047 ppm SO_2, 0.0048 mg/m³ acid mist and 0.88 mg/m³ dust) decreased body weight.	Oda et al., 1973
1,300 to 1,500	0.7 to 0.8	30 days	Mouse	No effect.	Nakajima et al., 1972
1,900 to 47,000	1 to 25	18 mo	Dog, rabbit, guinea pig, rat, hamster, mouse	No effect	Wagner et al., 1965
18,800	10	From birth to 62 days	Rat	Decreased body weight and length of pups.	Freeman et al., 1974b
18,800	10	Continuous, 90 days	Cynolmogus monkey	In combination with heat stress decreased body weight.	Coate and Badger, 1974
24,000	12.5	Continuous, 213 days	Rat	Decreased body weight.	Freeman and Haydon, 1964

pneumonia when exposed to such exhaust. An initial increase in the number of alveolar macrophages at the level of the terminal bronchioles, a proliferation of respiratory epithelium in the ducts, and a thickening of the alveolar septae were observed. Extramedullary hematopoiesis in rat livers resulted from high CO concentrations. Occasional degenerative changes in renal and hepatic tissue were also seen in these animals.

Lee et al. (1976) exposed lactating rats 24 hours/day for 7 days to auto emissions with and without catalytic conversion and with and without irradiation. Nitrogen dioxide concentrations in the unconverted auto exhaust were 8,650 and 9,780 $\mu g/m^3$ (4.6 and 5.2 ppm) for the irradiated and non-irradiated samples, respectively. For exhausts with catalytic conversion, NO_2 concentrations were 5,640 and 3,380 $\mu g/m^3$ (3 and 1.8 ppm) for irradiated and non-irradiated samples, respectively. Animals exposed to unconverted auto exhaust, whether irradiated or not, had a significant decrease in body weight by day 7 ($p < 0.001$). Exposure to converted and irradiated exhaust also produced a significant loss in weight ($p < 0.02$), but the presence of the converter was associated with less weight loss. Exposure to CO (575 mg/m^3; 500 ppm) did not significantly alter body weight. Animals exposed only to raw exhaust or 575 mg/m^3 (500 ppm) CO had hematocrit levels ($p < 0.001$) significantly elevated above controls by day 7. Exposure to raw exhaust enhanced serum lactate dehydrogenase activity (LDH), whereas CO had no such effect. Serum glutamate-oxaloacetate transaminase (SGOT) was not affected by any exposure regimen. Reduction of effects in converted exhaust-exposed groups was attributed to the decreased CO levels in the chamber.

Stupfel et al., (1973) in two separate experiments, exposed specific pathogen-free rats to auto exhaust fumes 6 to 8 hours/day, 5 days/week, for periods of 2.5 months to 2 years. The exhaust gas contained either 0.2 or 23 ppm NO_x, 0.07 or 0.37 percent CO_2, 58 mg/m^3 (50 ppm) CO and 0.1 or 2 ppm aldehydes. With low concentrations of NO_x, no biological effects were observed. When NO_x was increased to 23 ppm, body weight was reduced and spontaneous tumors and emphysema were increased. The heart rate and the QRS wave of the EKG were not affected. Sound avoidance reflexes were decreased.

Cooper et al. (1977) exposed rats 24 hours/day for 38 to 88 days to automobile exhaust with and without catalytic convertors. During three experiments, NO_2 levels were 564, 752, and 9,588 $\mu g/m^3$ (0.3, 0.4, and 5.1 ppm); nitric oxide (NO) levels were 8,733, 13,284, and 10,209 $\mu g/m^3$ (7.1, 10.8, and 8.3 ppm); and total hydrocarbon levels were 16, 14, and 50 ppm (methane). Spontaneous locomotor activity, as measured by standard running wheels, was 63, 54, and 64 percent of control values, respectively. The authors concluded that the suppression of activity was primarily related to either hydrocarbon or nitrogen oxide compounds of the exhaust. However, it should be noted that a 10-fold increase in NO_2 did not result in a further reduction in activity levels.

Murphy (1964) investigated the effects of a 4-hour exposure to irradiated or raw auto exhaust on pulmonary function of guinea pigs. Since irradiated exhaust produced more changes, subsequent studies were performed with irradiated exhaust maintained at equilibrium values or at cyclic values. For the equilibrium group, the following pollutant concentrations were measured: 2.42 ppm formaldehyde, 0.2 ppm acrolein, 0.8 ppm oxidant, 5,000 μg/m^3 (2.66 ppm) NO_2, and 300 ppm carbon monoxide (CO). For the "cyclic" exposure, concentrations varied: 1.3 to 1.9 ppm formaldehyde, 0.06 to 0.1 ppm acrolein, 0.56 to 0.95 ppm oxidant, 1,485 to 4,079 μg/m^3 (0.79 to 2.17 ppm) NO_2, and 150 to 250 ppm CO. Flow resistance was increased in both exposure groups with the equilibrium condition causing a more rapid and greater increase. During the first 1.5 hours of exposure, breathing frequency decreased, with the greater change occurring under equilibrium exposure conditions. For equilibrium exposure these values remained depressed, whereas in the cyclic group frequency increased. Increases in tidal volume were observed in both groups after 1.5 hours of exposure; in the cyclic group tidal volumes had decreased below control by 2.5 hours.

Beagles were exposed to auto exhaust and pollutant mixtures, 16 hours/ day, for 61 months. Pulmonary function studies were made at 18, 36, and 61 months of exposure (Lewis et al., 1974; Vaughan et al., 1969). The animals were allowed to recover for approximately 2 years (Gillespie et al., 1975) and were then examined again. Only those results related to NO_x will be described here. The results of this study have been described in full by several authors (Stera et al., eds., 1980). The high NO_2 group was exposed to 1,210 μg/m^3 (0.64 ppm) NO_2 plus 310 μg/m^3 (0.25 ppm) NO. The low NO_2 group was exposed to 270 μg/m^3 (0.14 ppm) NO_2 plus 2,050 μg/m^3 (1.67 ppm) NO. Vaughan et al.[168] reported no alterations in CO diffusing capacity, dynamic compliance, or total expiratory resistance to air flow after 18 months of exposure. By 36 months, (Lewis et al., 1974) analysis of variance indicated no significant changes; however, a significant number of animals exposed to high NO_2 and low NO had an abnormally ($p < 0.005$) low CO diffusing capacity. More changes were observed after 61 months of exposure (Lewis et al., 1974). In the dogs breathing the low NO_2 and high NO and raw auto exhaust, with and without SO_x, residual volume was increased ($p < 0.05$) compared to animals exposed to air or high NO_2 and low NO ($p < 0.05$). The common treatment factor causing this effect appeared to be the higher concentration of NO. Irradiated auto exhaust exposure increased ($p < 0.05$) the mean nitrogen washout values. A significant number of beagles exposed to high NO_2 and low NO had a lower mean carbon monoxide diffusing capacity/total lung capacity and a lower peak flow rate compared to control. A number of alterations in pulmonary function were found in other exposed groups. The authors attribute the results observed in the dogs exposed to high NO_2-low NO to an alteration of the alveolar capillary membrane.

After the 61-month exposure terminated, the animals were allowed to recover for 2 years before pulmonary function measurements were made again (Gillespie et al., 1975). In several instances, alterations occurred during this recovery period. In all pollutant-exposed dogs, total lung capacity was increased relative to the control group of animals. Those animals which received the NO_2 and NO mixtures experienced modest increases in inspiratory volume, vital capacity, and total lung capacity. Other groups of animals also had a number of changes.

Orthoefer et al., (1976) utilizing the same beagles exposed for 68 months, evaluated biochemical alterations 2.5 to 3 years after the animals had recovered. In groups exposed to irradiated auto exhaust with and without SO_x and high NO_2 with low NO, there was a rise ($p < 0.05$) in lung prolyl hydroxylase (the rate-limiting enzyme for collagen synthesis). A high correlation was seen between lung weights and hydroxyproline content in animals exposed to NO_x. No effects were observed in the hydroxyproline/total ninhydrin reactive material. Histological and morphological examinations revealed no significant differences in collagen content of exposed tissues. No difference was seen in the collagen/protein ratios between tissues.

Beagle lung morphology was evaluated by Hyde et al. (1978) 32 to 36 months after the 68-month exposure period terminated. Several morphometric parameters were affected, but only those relating to NO_x will be described here. In the high NO_2 group, there were increases ($p < 0.05$) in total lung capacity and right lung volume and decreases ($p < 0.05$) in the surface density of the alveoli and the volumetric density of parenchymal tissue. For the high NO group, there were no significant changes in the morphometric parameters examined. Alveoli were enlarged in both the high NO_2 and high NO groups. In the high NO_2 but not the high NO groups, there was increased ciliary loss without squamous metaplasia and nonciliated bronchiolar cell hyperplasia. In the high NO group, there were lesions in the interalveolar pores. In the most severely affected lungs of dogs in the high NO_2 group, morphological changes considered to be analogous to human centrilobular emphysema were present. Since these morphometric/ morphologic measurements were made after a 2.5 to 3 year holding period in clean air, it is not known whether these disease processes abated or progressed over the clean air period.

Coffin and Blommer (1967) investigated the effects of a 4-hour exposure to irradiated auto exhaust containing varying concentrations of NO_2, NO, CO, and oxidant on mice which were also challenged with _S. pyogenes_. Those mixtures containing greater than 752 $\mu g/m^3$ (0.4 ppm) NO_2 and 25 $\mu g/m^3$ (0.02 ppm) NO in the presence of 115 $\mu g/m^3$ (100 ppm) CO and between 0.52 and 0.67 ppm oxidant caused an increase in mortality ($p < 0.05$). Mixtures of NO_2 and NO at lower concentrations were not tested. Other pollutant combinations were also tested. Concentrations between 376 and 1,500 $\mu g/m^3$ (0.2 and 0.8 ppm) NO_2, 0.15 and 0.48 ppm oxidant, and 29 and 115 $\mu g/m^3$ (25 and 100 ppm) CO were found to increase mortality in the infectivity model.

Using the infectivity model in which the mortality of pollutant-exposed mice challenged with _S. pyogenes_ is measured, Ehrlich et al. (1977) investigated the effects of combinations of O_3 and NO_2. Mice were exposed for 3 hours to various concentrations of the gases alone and in combination. The lowest concentrations causing a significant enhancement of mortality were a mixture of 98 $\mu g/m^3$ (0.05 ppm) O_3 and 3,760 $\mu g/m^3$ (2 ppm) NO_2. The effect was additive. This concentration of O_3 alone caused no significant change; 3,760 $\mu g/m^3$ (2 ppm) NO_2, however, resulted in a significant enhancement of mortality. Multiple 3-hour daily exposures also were tested. With the mixture of 98 $\mu g/m^3$ (0.05 ppm) O_3 and 3,760 $\mu g/m^3$ (2 ppm) NO_2, excess mortalities were evident after five daily exposures. At 196 μg/m^3 (0.1 ppm) O_3 plus 2,800 μg/m^3 (1.5 ppm) NO_2, there was a significant effect when the mice were examined after 20 daily exposures, but not after 10. In the latter case, the author attributed the effect to the presence of O_3; in the former case, the author suggested a synergistic relationship. When mice were exposed to a combination of 6,580 μg/m^3 (3.5 ppm) NO_2 and 980 μg/m^3 (0.5 ppm) O_3 for 3 hours, followed 1 hour later by challenge with *S. pyogenes*, bacterial clearance from the lung was reduced. Control mice cleared 50 percent bacteria from the lungs in 81 minutes. Exposed animals took 131 minutes to clear the same amount of bacteria. When the NO_2 concentration was reduced to 3,760 μg/m^3 (2 ppm) and the initial O_3 concentration was maintained, no distinguishable effect on bacterial clearance was observed.

Ehrlich et al. (1979) used another exposure regimen of NO_2-O_3 combinations to evaluate effects with the streptococcal infectivity model. For 1, 2, 3, and 6 months, mice were exposed continuously to 188 μg/m^3 (0.1 ppm) NO_2 on which were superimposed peaks (3 hr/day, 5 days/wk) of either 940 μg/m^3 (0.5 ppm) NO_2 or 940 μg/m^3 (0.5 ppm) NO_2 plus 196 μg/m^3 (0.1 ppm) O_3. Other groups received the peak exposures only. After the indicated exposure period, mice received *S. pyogenes* and were observed for mortality over a 14-day period in clean air. For the 1- or 2-month exposures the only significant ($p < 0.05$) change was a decrease in mortality after 1 mo. of peak exposure to the $NO_2 + O_3$ mixture. After 3 mo., the peak exposures to NO_2 only or $NO_2 + O_3$ increased ($p < 0.05$) mortality. At the same time, there was a decrease ($p < 0.05$) in mortality in the group exposed to a baseline of NO_2 with peaks of either NO_2 or $NO_2 + O_3$. However, mortality increased ($p < 0.05$) after 6 mo exposure to NO_2-O_3 peaks, with or without the baseline of NO_2. In another set of experiments using identical exposure regimens, the mice were returned to their respective pollutant exposures over the 14-day observation period following bacterial challenge. In these studies, the increased susceptibility to infection occurred earlier compared to those animals held in clean air for the 14 day period. Generally in these animals, the most changes occurred in the group receiving the $NO_2 + O_3$ peak, and there was an increased response as exposure length increased. When the peak of $NO_2 + O_3$ was superimposed on a baseline of NO_2, the response was reduced, although it did increase with time. The effects in the peak of NO_2 group were roughly equivalent to those in the group receiving a baseline of NO_2 with an NO_2 peak superimposed. These exposure regimens also affected alveolar macrophages. For example, the most extensive effect occurred after a 3 mo. exposure to the peak of $O_3 + NO_2$. There were decreases ($p < 0.05$) in the number of recoverable alveolar macrophages, their percent viability and percent phagocytosis. Lung morphological changes in infected mice were also observed using scanning electron microscopy. Marked changes were observed after 3 mo. of exposure to a baseline of NO_2 or air on which were superimposed peaks of $NO_2 + O_3$. Alveolar pores were greatly enlarged in some areas. Adjacent to these areas, alveolar walls were thickened and fused with smaller pores. Peak exposures to NO_2 only caused less extensive effects.

Goldstein et al. (1974) found that mixtures of NO_2 and O_3 would decrease bactericidal activity of the lungs. Mice received S. aureus aerosols before a 4-hour pollutant exposure. Reductions ($p < 0.01$) in bactericidal activity of 36.9 and 111 percent were seen after exposure to 7,520 µg/m^3 (4 ppm) NO_2 plus 600 µg/m^3 (0.36 ppm) O_3, and after 12,860 µg/m^3 (6.84 ppm) NO_2 plus 760 µg/m^3 (0.39 ppm) O_3 respectively. Lower gas concentrations had no effect. The protocol was then changed so that mice were exposed to pollutants for 17 hours before S. aureus challenge. Bactericidal activity was generally more impaired at higher gas concentrations. The lowest gas concentrations to cause a significant effect ($p < 0.05$) were 4,320 µg/m^3 (2.3 ppm) NO_2 plus 390 µg/m^3 (0.2 ppm) O_3 which decreased bactericidal activity 13 percent. However, decreases in pulmonary bacterial deposition were observed at these concentrations. It is the authors' contention that this latter effect is due to ventilatory defects induced by O_3 alone, whereas the reduction in bactericidal function is equivalent to the injury that would be expected from each individual gas.

Furiosi et al. (1973) exposed monkeys and rats to aerosols containing 330 µg/m^3 (0.14 ppm) NaCl and 3,760 µg/m^3 (2 ppm) NO_2 continuously for 14 months in order to delineate the effect of particulate aerosols on NO_2 toxicity. Of the total NaCl aerosolized, only 5 µg/m^3 (< 0.001 ppm) had a particle size between 5 and 10.3 microns, with the remainder being smaller. NaCl aerosol alone had no effect on the experimental animals. Following 14 months exposure, the bronchiolar epithelium was hypertrophic to similar degrees in monkeys exposed to NO_2 or NO_2 in the presence of NaCl. With only half the concentration of NaCl and NO_2, rats exposed to these agents revealed marginal results. Animals exposed to NO_2 with and without NaCl developed polycythemia with reductions in mean corpuscular volumes although mean corpuscular hemoglobin concentration was normal.

Kosmider et al. (1973a) exposed guinea pigs 8 hours/day for 6 months to 1 ppm oxides of nitrogen ($NO_2 + N_2O_4$) or 1 ppm oxides of nitrogen ($NO_2 + N_2O_4$) plus a somewhat larger quantity of ammonia. The oxides of nitrogen alone decreased animal body weight gain over the 6-month period; 62 grams versus 395 grams for controls and 120 grams for NO_x plus ammonia. Reduction in total serum protein was observed with a marked decrease in the levels of albumin, alpha-2 and gamma globulins and with an increase in alpha-1 and beta fractions. Higher incidences of spontaneous infections also were observed. Disorders in acid-base balance were reported. Increased appearance of urobilinogen, acid mucopolysaccharides, and hydroxyproline was observed. Hemorrhage and emphysematous-like conditions were noted in the lungs of NO_x-exposed animals. Emik et al. (1971) reported that alkaline phosphatase activity decreased in the lungs of rats exposed 2.5 years to ambient air containing approximately 36 µg/m^3 (0.019 ppm) NO_2, 0.011 ppm NO, O_3, PAN, etc.

Antweiler and Brockhaus (1976) exposed female guinea pigs 6 days/week for 6 months to 10,000 µg/m^3 (3.8 ppm) SO_2, 10,000 µg/m^3 (5.3 ppm) NO_2, or a combination of the two pollutants. After 6 months of exposure, there were no effects on respiratory frequency, flow rate, or minute volume, nor were there increases in mortality or difference in weight gain between treated and control animals.

Shalamberidze and Tsereteli (1971) reported on the effects of low concentrations of SO_2 and NO_2 on the estrous cycle and reproductive functions of rats. Female albino rats were exposed 12 hours/day for 3 months to one of the following: (1) 2,360 $\mu g/m^3$ (1.3 ppm) NO_2; (2) 5,000 $\mu g/m^3$ (1.9 ppm) SO_2; (3) 160 $\mu g/m^3$ (0.06 ppm) SO_2 ; (4) 130 $\mu g/m^3$ (0.07 ppm) NO_2; (5) 2,500 $\mu g/m^3$ (0.95 ppm) SO_2 and 1,130 $\mu g/m^3$ (0.6 ppm) NO_2. Low concentrations of SO_2 and NO_2 did not affect the rats' estrous cycle or induce morphological changes in reproductive organs. Exposure to high concentrations did alter the estrous cycle. Estrus was less frequent, more prolonged, and occurred at lengthened intervals. At 7 months after exposure, the estrual indices returned to normal. Morphologically, changes included a depletion of glandular epithelium in the uterus as well as a depletion of thyroid connective tissue between follicles with a mild cellular degeneration of the adrenals, ovaries, and uterus. The number of ovarian primordial follicles was also decreased. Long-term exposure had no effect on the rats' ability to conceive, although litter size and average weight of progeny were significantly reduced ($p < 0.001$).

Oda et al. (1975a; 1975b) exposed female mice and male rats and rabbits for 1 hour to 13,040 $\mu g/m^3$ (10.6 ppm) NO containing 1,500 $\mu g/m^3$ (0.8 ppm) NO_2. Shortly after NO exposure, mice and rats had increased nitrosylhemoglobin (NOHb). Production of NOHb was proportional to the concentration of NO. Within 20 minutes equilibrium was reached, with NOHb being 0.13 percent of total hemoglobin. NOHb levels declined rapidly when mice were placed in clean air for 1 hour. NOHb had a half-life of 10 minutes. NOHb was not detected in rabbits until reduced *in vitro* with sodium dithionate.

14.4 NITRIC OXIDE

The toxicological data base of nitric oxide (NO) is not extensive and most of it has been done in recent years. (See Table 14-19.)

Azoulay et al. (1977) exposed rats continuously for 6 weeks to 2,460 $\mu g/m^3$ (2 ppm) NO. Following exposure, no significant differences were seen in blood oxygen saturation, pH, oxygen combining capacity, 2,3-diphosphoglycerate, ATP, glucose, lactate, hemoglobin concentration, hematocrit, and red blood cell count. Methemoglobin was not detected. No striking histological changes were found in the lungs until 2 weeks of exposure. At this time, 3 of 4 exposed and 1 of 4 controls appeared to show inflammatory changes including cellular infiltration of alveolar cells. Inflammatory changes, including cellular infiltration of alveolar walls and areas of intra-alveolar edema, were observed. Following 3 weeks of exposure to NO, animals evidenced some emphysemic-like changes with increased incidence until 6 weeks of exposure. Hugod (1979) exposed rabbits continuously for 6 days to 52,890 $\mu g/m^3$ (43 ppm) NO and 6,768 $\mu g/m^3$ (3.6 ppm) NO_2. No significant effect on lung morphology was observed following exposure.

Arnold et al. (1977b) and Braughler et al. (1979) exposed various tissues from male rats *in vitro* to 25 to 250 µl NO gas for 10 seconds. The activity of guanylate cyclase (GC) (an enzyme that forms cyclic GMP, an important biochemical regulator of cells) in various tissues

TABLE 14-19. NITRIC OXIDE

Pollutant	Pollutant Concentration µg/m³	ppm	Exposure	Species	Effect	Reference
NO	2,460	2.0	Continuous, 6 wk	Rat	Cellular infiltration of alveolar walls and areas of intra-alveolar edema observed after 2 wk. After 3-wk exposure, emphysema-like changes observed until 6 wk. Methemoglobin undetected. No differences in blood oxygen saturation, pH, oxygen combining capacity, ATP, 2,3-diphosphoglycerate, glucose, lactate, hemoglobin concentration, hematocrit, or RBC count.	Azoulay et al., 1977
NO	9,470	7.7	8 hr/day, 120 days	Guinea pig	Decreased blood sodium, magnesium, and chloride; decreased Zn and Mg in brain and liver. Increased blood calcium and urinary excretion Mg.	Kosmider and Chorazy, 1975
NO + NO_2	13,040 + 1,500	10.6 + 0.8	1 hr	Mice, female; Rat, male; Rabbit, male	Mice and rats showed increased nitrosyl hemoglobin (NOHb); NOHb related to concentration of NO. By 20 minutes equilibrated, 0.13% total hemoglobin. NOHb half-life 10 minutes. No NOHb produced in rabbits exposed to NO until sodium dithionate added to blood.	Oda et al., 1975a Oda et al., 1975b
NO	14,800	12	5 hr	Mouse	Edema and dilation of vessels in submucosal tissue of trachea. Congested alveoli. 24 hr later proliferation of tracheal mucosal epithelium. Anomalies in CNS, heart and cell metabolism.	Udai et al., 1973
NO	19,700 to 61,500	16 to 50	4 hr	Guinea pig	No significant alteration in respiratory rate or tidal volume.	Murphy et al., 1964
NO+ NO_2	52,890 + 6,768	43 + 3.6	Continuous, 6 days	Rabbit	No effect on lung morphology.	Hugod, 1979

was increased in proportion to the dose of NO. The levels of this enzyme were increased 19- to 33-fold in supernatants of liver, lung, tracheal smooth muscle, heart, kidney, cerebral cortex, and cerebellum with increases of 5- to 14-fold in supernatant of skeletal muscle, spleen, intestinal muscle, adrenal, and epididymal fat pads. Following NO activation, GC activity decreased with a half-life of 3 to 4 hours at 4°C. When tissue was re-exposed, the GC activity was reactivated. Braughler et al. (1979) also found that exposure of rat liver fractions to 165 µl NO increased GC activity, but purification of the enzyme resulted in an apparent loss of enzyme activation by NO. Restoration of this activity could be accomplished with the addition of dithiothreitol, methemoglobin, BSA, or sucrose. Sodium nitrite increased GC activity as well. Nitric oxide increased cyclic GMP (cGMP) but had no effect on cyclic AMP (cAMP). Craven and DeRubertis (1978) exposed rat liver fractions to NO at the rate of 1 ml/min for 2 seconds and found similar increased activity in liver GC. They also found that a nitrosyl-heme complex was formed during exposure.

Kosmider and Chorazy (1975) reported that following the exposure of guinea pigs to 9,470 $\mu g/m^3$ (7.7 ppm) NO 8 hours/day for 120 days, blood sodium, magnesium, and chloride were reduced with a significant rise in calcium. Liver and brain levels of magnesium and zinc were reduced while there was an enhanced urinary excretion of magnesium.

14.5 NITRIC ACID AND NITRATES

Gray et al. (1952) conducted some of the earliest experiments investigating inhalation toxicity for rats, guinea pigs, and mice exposed to NO_2 generated from nitric acid (HNO_3). (See Table 14-20.) No measurement of aerosol particle size was made. Results were expressed as evidence of NO_2 toxicity, whereas the experimental method could not adequately distinguish the difference between NO_2 and HNO_3 effects. Concentrations of 17,000 and 26,000 $\mu g/m^3$ (9 and 14 ppm) NO_2 administered 4 hours/day, 5 days/week for 6 weeks produced lung pathology. When exposure concentration was reduced to 9,400 $\mu g/m^3$ (5 ppm), no lesions were observed.

Gardiner and Schanker (1976) investigated the effects of nitric acid-induced lung damage on the absorption of drugs from the lungs. Rats were given an intratracheal injection of 1 percent nitric acid solution (0.15 ml), and drug absorption rates from treated versus control lungs were measured with time. One day following nitric acid exposure, there was significant bronchiolar inflammation with inflammatory cell infiltration. The bronchiolar epithelium lost its normal scalloped appearance and tended to have an increase in cellular cytoplasm. Alveolar septae adjacent to inflamed bronchioles appeared broadened by enlarged or swollen alveolar cells. Wet and dry weights of the exposed lungs did not differ. Treatment with nitric acid enhanced the pulmonary absorption rates (20 percent) of p-aminohippuric acid, procaineamide ethobromide, procaineamide, and mannitol.

Sackner et al. (1976) exposed dogs for 7.5 minutes to an aerosol, with particles less than 1 µm in diameter, containing 740 and 4,000 $\mu g/m^3$ (0.1 and 1 percent) of sodium nitrate in order to observe potential effects on cardiopulmonary function. Sodium nitrate at either concentration had no effect on functional residual capacity, static lung compliance, or total respiratory resistance.

TABLE 14-20. NITRIC ACID AND NITRATES

Pollutant	Pollutant concentration	Exposure	Species	Effect	Reference
NO_2 generated from HNO_3	9,400, 17,000 or 26,000 μg/m^3 (5, 9 or 14 ppm)	4 hr/day, 5 days/wk, 6 wk	Rat, mouse, guinea pig	No lesions at 9,400 μg/m^3 (5 ppm). Higher concentrations, increased lung pathology.	Gray et al., 1952
HNO_3	1% solution (0.15 ml)	Intra-tracheal injection	Rat	24 hr post-injection increased inflammation of bronchioles; epithelium lost normal scalloped appearance. Increased cytoplasm in epithelium. Inflamed alveolar septae. No difference in lung wet and dry weight. Enhanced pulmonary absorption rates of p-aminohippuric acid, procaineamide ethobromide, procaineamide and mannitol.	Gardiner and Schanker, 1976
Sodium nitrate	0.1 and 1.0% at 740 and 4,000 μg/m^3	7.5 min	Dog	No effect on pulmonary function.	Sackner et al., 1976
Ammonium nitrate	100 mM	30 min	Guinea pig	Accounted for 58% of total histamine release. Ammonium sulfate released.	Charles and Menzel, 1975
$Pb(NO_3)_2$ $Ca(NO_3)_2$ $NaNO_3$ KNO_3 NH_4NO_3	2,000 μg/m^3 2,800 μg/m^3 3,100 μg/m^3 4,300 μg/m^3 4,500 μg/m^3	3 hr	Mouse	After 14 days of observation, no effect on mortality following challenge with *S. pyogenes*.	Ehrlich, 1979
$ZnNO_3$	1,250 μg/m^3			Increased mortality.	

Charles and Menzel (1975) incubated 100 mM ammonium nitrate with guinea pig lung fragments for 30 minutes and measured the release of histamine. Ammonium nitrate released 58.1 percent of the total histamine.

Ehrlich (1979) investigated the effect of nitrates on resistance to respiratory infection in mice. The animals were exposed to the compounds for 3 hr, after which they were challenged with a viable aerosol of S. pyogenes, and mortality was determined after a 14-day observation period. No significant effects were observed at the following maximal concentrations tested: 2,000 μg/m^3 $Pb(NO_3)_2$, 2,800 μg/m^3 $Ca(NO_3)_2$, 3,100 μg/m^3 $NaNO_3$, 4,300 μg/m^3 KNO_3, and 4,500 μg/m^3 NH_4NO_3. However, zinc nitrate increased mortality in a significant linear concentration-related fashion with about 1,250 μg/m^3 causing a 20 percent increase in mortality. This result was similar to that of zinc sulfate. Thus, it appeared that the effect was more due to the cation than the anion.

14.6 N-NITROSO COMPOUNDS

Reviews of the acute toxicity, carcinogenicity, mutagenicity, and detection of nitrosamines have appeared recently. (Magee et al., 1977; Montesano and Bartsch, 1976; U.S. Environmental Protection Agency, 1977) These compounds and nitrosamides are highly toxic and potent carcinogens. N-Nitrosamines require metabolic activation to their mutagenic and carcinogenic active intermediates and are most toxic to the liver and kidneys. Nitrosamides, on the other hand, tend to decompose at physiological pH, probably to active intermediates similar to those produced from nitrosamines, and thus often act locally.

The detection of nitrosamines in food and water had promoted a broader search for their presence in the environment. Evidence for the presence of dimethylnitrosamine in air has been reviewed in the Scientific and Technical Assessment Report on Nitrosamines (U.S. Environmental Protection Agency, 1977). Henschler and Ross (1966) investigated the possible formation of nitrosamines from tissue amines exposed to NO_2. Mice were exposed intermittently to 75,200 μg/m^3 (40 ppm) NO_2 for 38 hours every tenth day for periods up to 1.5 years. No lung tumors were found in exposed animals. In fact, when compared to controls, there was a slight inhibition of the formation of lung adenomas and spontaneous skin fibro-adenomas. The formation of nitrosamines by reaction of amines with nitrogen oxides has been observed in the laboratory but not in the atmosphere (Challis, 1977; Gehlert and Rolle, 1977). Nitrosamines would not be expected to accumulate to any great extent in ambient air because they are readily decomposed by sunlight (Chow, 1973; Polo and Chow, 1976). Kaut (1970) examined the lungs of rats exposed for 3 hours to mixtures of 10,000 to 500,000 μg/m^3 nitrogen oxides for the presence of nitroso compounds. None of the compounds were found *in vivo*, whereas they were found *in vitro* when lung homogenates were exposed to high concentrations of nitrogen oxides (15 percent).

The nitrosamines are acutely toxic with a single oral dose of 27,000 to 41,000 μg/m^3 being the LD50 for dimethylnitrosamine to the rat. Diethylnitrosamine, another compound detected in the air, is much less toxic with an LD50 of 216,000 μg/m^3 (Heath and Magee, 1962). Inhalation toxicity has not been reported. Industrial use of nitrosamines as solvents suggest that they are rapidly absorbed and exert their toxic action equally well on inhalation and ingestion. These compounds were acutely toxic to every animal species tested and were also poisonous to humans (Barnes and Magee, 1954; Freund, 1937).

Nitrosamines have caused hepatotoxicity, including the formation of "blood cysts", which are the necrotic areas of parenchyma filled with recently extravasated erythrocytes, and central and sublobular hepatic necrosis. Renal tubule damage is the dominant feature of the kidney damage. Dimethylnitrosamine produces venous occlusions in the liver (McLean et al., 1965). Ultrastructural changes in the liver after diethyl- and dimethylnitrosamine include separations of the fibrillar and granular components of the hepatocyte and the formation of electron dense plaques at the periphery of the nucleolus (Svoboda and Higginson, 1968). Lysosomal alterations occurred within 35 minutes of exposure to dimethylnitrosamine and reached a maximum at 12 hours (Svoboda and Higginson, 1968).

Nitrosamines and nitrosamides (N-nitroso compounds) have induced tumors in a wide variety of organs of experimental animals, often at organs distant from the site of administration. Single or repeated inhalation exposures to dimethylnitrosamine resulted in tumors of the nasal cavities and kidneys (Druckrey et al., 1967). N-Nitrosoheptamethyleneimine produced squamous neoplasia of the lung in rats, histologically similar to human lung cancer (Lijinsky et al., 1969).

Many N-nitroso compounds are mutagenic if assayed by the appropriate system. Bacteria, in general, are not capable of activating nitrosamines, and such assay systems therefore require supplementations with animal-derived activating enzymes to detect the mutagenicity of these compounds. Nitrosamides, which will spontaneously decompose in the bacteriological medium, do not require enzyme activation. More than 20 N-nitroso compounds have been shown to be mutagenic in microbial systems (McCann et al., 1975).

N-Nitroso compounds are also teratogens. The nitrosamide, N-nitrosoethylurea, given to rats on the 12th day of gestation (Napalkov and Alexandrov, 1968) or N-nitrosomethyl-urea given on the 13th or 14th (Von Kreybig, 1965) can cause fetal death and resorption and, for the progeny which do reach term, a variety of malformations.

The N-Nitroso compounds, therefore, are an important class of chemical carcinogens for the following reasons:

Most vital tissues are susceptible to the carcinogenic action of this class of compounds. Bone can be included in this list, based on the recent finding that 1-(2-hydroethyl)-1-nitrosourea induced osteogenic osteosarcomas or chondrosarcomas of the lower vertebrae in rats (Pelfrene et al., 1967).

In several instances, a single exposure to neonatal animals has induced tumors as the animals reached adulthood (Druckrey et al., 1963; Magee and Barnes, 1959). Single-exposure induction of tumors can also occur in adult rats that are pregnant (Druckrey et al., 1967) or recovering from a partial hepatectomy (Craddock, 1971).

N-Nitroso compounds can induce cancers transplacentally. Brain and spinal cord tumors (Ivankovic and Druckrey, 1968) and renal tumors (Wrba et al., 1967) were found in progeny of pregnant rats treated with N-nitrosoethylurea. Diethylnitrosamine has transplacentally induced tracheal papillomas in rats (Mohr et al., 1966).

Small amounts of carcinogenic nitrosamines have been detected in some samples of urban air (Fine et al., 1976) and in the food supply (Crosby and Sawyer, 1976).

N-Nitroso compounds could present a hazard, not only because people are exposed to these compounds but also because they can be produced *in vivo*, at least in experimental animals. Nitrosation reactions are catalyzed by acid and hence should occur preferentially in the stomach. Their reactions have been induced in animals by feeding nitrite with amines or amides (Mirvish, 1972; 1977; Rounbehler et al., 1977). Also, under these situations acute toxic effects have been observed and N-nitroso compounds have been detected by chemical analysis (Mirvish, 1975).

A recent report by Iqbal et al. (1980) demonstrates *in vivo* biosynthesis of N-nitrosomorpholine from mice exposed to NO_2 and morpholine. This is the first and only report of a direct link between NO_2 exposure and nitrosamine formation *in vivo*. N-nitrosomorpholine was detected in animals with as low as 376 $\mu g/m^3$ (0.2 ppm) NO_2 exposure for 4 hours. After 4 hours of exposure at 94,000 $\mu g/m^3$ (50 ppm) NO_2, a maximum of 2230 ng N-nitrosomorpholine/mouse was detected. These quantities are extremely small, and it is not known what the biological significance (in terms of mutation or cancer) is to this exposure. However, the possibility of low level exposure to NO_2 and concomitant biosynthesis of nitrosamines indicates a potential health hazard. This area of investigation requires additional work in order to quantitate this potential health hazard to man (Iqbal et al., 1980).

14.7 SUMMARY

The biological effects of nitrogen oxides have been examined in a number of animal species. Thus far, the most toxic among these is nitrogen dioxide (NO_2). A summary of the research results observed at 18,800 $\mu g/m^3$ (10.0 ppm) NO_2 or less is set forth in Table 14-21. While mechanisms of action can be studied by exposures at or above 18,800 $\mu g/m^3$ (10.0 ppm) NO_2, this concentration is judged to be the maximum at which animal studies provide relevant data to estimating the human health effects of ambient or near ambient concentrations of NO_2.

An unusual aspect of the toxicity of NO_2 is a delay between exposure and effect. This temporal sequence is inherent in understanding the toxicity of NO_2 and has important implications for the effects of both short-term and long-term exposures to this air pollutant. Despite the differences in NO_2 sensitivity among animal species and the many different endpoints of toxicity, illustrated in Figure 14-4. A composite has been drawn from these different studies to illustrate the relationship with time following a single short-term exposure of 4 hours or less. These results are drawn primarily from a single species, the rat. A similar sequence could be drawn for other species. It is likely that this sequence is the

TABLE 14-21. SUMMARY OF EFFECTS OF NO_2 IN ANIMALS AT CONCENTRATIONS OF 10 ppm OR LESS

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
188	0.1 spike 2 x daily with 0.05 baseline	Continuous, 15 days	Mouse	No effect on S. pyogenes infectivity	Gardner, et al., 1981
188 + 2 hr daily spike of 1,880	0.1 + 2 hr daily spike of 1	Continuous 6 mo	Mouse	Emphysematous alterations	Port, et al., 1977
376	0.2	3 hrs	Rat	Inhibition of conversion of PGE_2 to its metabolite. No effect on PGE_2 uptake or efflux	Menzel, 1980
470	0.25	4 hr/day 5 days/wk 24 or 36 days	Rabbit	Swollen collagen fibers in lung	Buell, 1970
470	0.25	3 hr/day up to 3 d	Mouse	Increased pentobarbital sleeping time in female mice after 1 day. No effects after 2 or 3 days.	Miller, et al., 1980
560-940	0.3-0.5	Continuous 3 mo	Mouse	NO_2 + influenza virus caused a high incidence of adenomatous proliferation of peripheral and bronchial epithelial cells	Motomiya, et al., 1973

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
600	0.32	3 mo	Rat	Decreased conditioned reflexes	Yakimchuk and Chelikanov, 1958
680	0.36	7 days	Guinea Pig	Increased erythrocyte D-2, 3-diphosphoglycerate	Mersch, et al., 1973
740	0.4	4 hr/day 7 days	Guinea Pig	Increase in lung acid phosphatase	Sherwin, et al., 1974
740	0.4	Continuous 1 wk	Guinea Pig	Increase in protein of lung lavage in vitamin C deficient animals	Sherwin and Carlson, 1973
740	0.4	Continuous 2 wk	Guinea Pig	No effect on protein or lipid in lung lavage in vitamin C deficient animals.	Selgrade, et al., 1981
940	0.5	6,18 or 24 hr/day 12 mo	Mouse	Morphological effects in alveoli	Blair, et al., 1969
940	0.5	8 hr/day 7 days	Guinea Pig	Increase in serum LDH, CPK, SGOT, SGPT and cholinesterase, and lung and plasma lysozyme. Decrease in erythrocyte GSH peroxidase. No change in lung GSH peroxidase.	Donovan, et al., 1976; Menzel, et al., 1977
940	0.5	Continuous 14 days	Guinea Pig	Albumin and globulins in urine	Sherwin and Layfield, 1974

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
1,000 NO_x (mostly NO_2)		8 hr/day 180 days	Guinea Pig	Nitrates and nitrites in urine; slight increase in serum cholesterol; decrease in total serum lipids; hepatic edema; increase in urinary Mg and decrease in liver and brain Mg	Kosmider, 1975
940	0.5	Continuous 30-45 days	Mouse	Morphological alterations of tracheal mucosa and cilia	Hattori, et al., 1972; (Nakajima), et al., 1969
940	0.5	5 days/wk 7 wk	Mouse	Increase of injected horseradish peroxidase in lung	Sherwin, et al., 1977
940	0.5	Continuous 8 hr/day 4 mo	Guinea Pig	Decrease in plasma cholinesterase; erythrocyte or lung GSH peroxidase unchanged. Increase in lung acid phosphatase and plasma and lung lysozyme	Donovan, et al., 1976; Menzel, et al., 1977
940 or 3,760	0.5 or 2	Continuous with 1 hr peaks of 2 ppm 5 days/wk	Mouse	Morphological alterations of alveolar macrophages; decreased serum neutralizing antibody to influenza virus immunization; changes in serum immunoglobulins	Aranyi, et al., 1976; Ehrlich, et al., 1975
940	0.5	Continuous 12 mo	Mouse	At 10 days: damage to clara cells and cilia and alveolar edema. At 35-40 days: bronchial hyperplasia. At 6 mo: fibrosis. At 12 mo: bronchial hyperplasia	Hattori, 1973; Hattori and Takemura, 1974

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
940 or 1,880	0.5 or 1	Continuous 1 yr, 5 mo	Mouse	No increase in lipofuscin or glutathione peroxidase	Ayaz and Csallany, 1977
940 or 1,880	0.5 or 1	Continuous 1 yr, 6 mo	Mouse	Growth reduced; vitamin E improved growth	Csallany, 1975
940	0.5	Continuous or intermittent (7-8 hr/day)	Mouse	Increased susceptibility to *K. pneumoniae* after 90 days continuous or 180 days intermittent exposure	Ehrlich and Henry, 1968
1,000	0.53	180 days 8 hr/day	Guinea Pig	Alterations in several serum enzymes	Drozdz, et al., 1975
1,030 - 3,000	0.55 - 1.6	Continuous 5 wk	Mouse	Cilia damaged; increased mucus secretion	Miyoshi, et al., 1973
1,500	0.8	Continuous 2.75 yr	Rat	Increase in respiratory rate	Haydon, et al., 1956; Freeman, et al., 1966
1,880	1	Continuous 3 days	Guinea Pig	Increase protein and lipid content of lavage fluid in vitamin C- depleted but not normal	Selgrade, et al., 1981
1,880	1	Continuous 2 wk	Rabbit	Decrease in lung lecithin synthesis after 1 wk; less marked depression after 2 wk	Seto, et al., 1975

TABLE 14-21. (continued)

Concentration of NO_2		Time of exposure	Species	Summary of effects	References
μg/m³	ppm				
1,880 2,820	1-1.5	Continuous 1 mo	Mouse	Hypertrophy of bronchiolar epithelium after 1-3 mo. After recovery from exposure, lymphocyte infiltration	Chen, et al., 1972
1,880	1	Continuous 6 mo	Guinea Pig	Inhibition of protein synthesis; decrease in body weight, total serum proteins, and immunoglobulins	Kosmider, et al., 1973a
1,880	1	Continuous 493 days	Monkey	Immunization with monkey-adapted influenza virus. Increased serum neutralizing antibody titers at 93 days of exposure. No change in hemagglutination inhibition titers; no effect on hematocrit or hemoglobin; increased leukocytes in blood. Slight emphysema and thickened bronchial and bronchiolar epithelium in virus-challenged monkeys	Fenters, et al., 1973
2,000	1.1	8 hr/day 180 days	Guinea Pig	Plasma and liver changes: decrease in albumin, seromucoid, cholinesterase, alanine and aspartate transaminases; increase in alpha and $beta_2$ immunoglobulins	Drozdz, et al., 1976
940 + 2 x daily spikes of 1880	0.5 + 2 x daily spikes of 1	Continuous 15 days	Mouse	Increase in mortality due to S. pyogenes	Gardner, et al., 1981

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
2,300 + 2 x daily spikes of 4,700	1.2 + 2 x daily spikes of 2.5	Continuous 15 days	Mouse	Increase in mortality due to S. pyogenes	Gardner et al., 1981
2,360	1.26	12 hr/day 3 mo	Rat, prior to breeding	No effect on fertility; decrease in litter size and neonatal weight; no teratogenic effects	Goldstein et al., 1973
2,400-5,200	1.3-3	2 hr/day 15 & 17 wk	Rabbit	Increased leukocytes in blood with decreased phagocytosis; decreased number of erythrocytes	Blair et al., 1969
2,800	1.5	Continuous or intermittent (7 hr/day, 7 days/wk)	Mouse	After 1 wk, increased susceptibility to S. pyogenes aerosol greater in continuous exposure group. After 2 wk, no significant difference between modes of exposure	Gardner et al., 1979
2,800 + 8,100 spike	1.5 + 4.5 spike	Continuous 62 hrs before & 18 hr after a spike for 1,3.5 or 7 hr	Mouse	Increased susceptibility to S. pyogenes aerosol after 3.5 or 7 hr single spike when bacterial challenge was delayed 18 hrs after spike	Gardner et al., 1981
2,800 + 8,100 spike	1.5 + 4.5	14 d; 2 daily 1 hr spike x 5 d/wk x 2 wk	Mouse	Increased susceptibility to S. pyogenes aerosol	Gardner et al., 1981
5,600	3	3 hr	Mouse	Increased susceptibility to S. pyogenes aerosol in mice exercising compared to those not exercising	Illing et al., 1980
3,760	2	3 hr	Mouse	Increased susceptibility to S. pyogenes aerosol	Ehrlich et al., 1977

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
3,760	2	Continuous 1-3 wk	Guinea Pig	Increase in number of lactic acid dehydrogenase positive lung cells (presumably Type II cells) with time of exposure	Sherwin, et al., 1972
3,760	2	Continuous 3 wk	Guinea Pig	Type II cell hypertrophy	Sherwin, et al., 1973
3,760	2	Continuous 43 days	Rat	Between 72 hr - 7 days increasing loss of cilia and focal hyperplasia; by 14 days, cilia regenerated and recovery was evident at 21 days	Stephens, et al., 1972
3,800	2	Continuous 14 mo.	Rat Monkey	Polycythemia with or without NaCl. Hypertrophy of bronchiolar epithelium	Furiosi, et al., 1973
3,800	2	Continuous 2 yr.	Rat	Increase in respiratory rate; no change in resistance or dynamic compliance	Freeman, et al., 1968c
4,280	2.3	17 hr	Mouse	Decreased pulmonary bactericidal activity (no measurable effect at 1 ppm x 17 hr. or 3.8 ppm x 4 hr.)	Goldstein, et al., 1973b
5,000	2.7	8 wk	Rat	Decreased body weight	Kaut, et al., 1966
5,450	2.9	Continuous 20 days	Rat	Decrease in linoleic and linolenic acid of lung lavage fluid	Menzel, et al., 1972

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	ppm	Time of exposure	Species	Summary of effects	References
5,450	2.9	24 hr/day 5 days/wk 9 mo.	Rat	Decrease in lung compliance and volume; increased lung weight and decreased total lung lipid; decreased saturated fatty acid content of lung lavage fluid and tissue; increased surface tension of lung lavage fluid	Arner and Rhoades, 1973
5,640	3	4 hr/day 4 days	Monkey	Thickening of basal laminar and alveolar walls; interstitial collagen	Bils, 1976
5,640	3	Continuous 3 days	Guinea Pig	Increased protein and lipid content of lavage fluid in vitamin C-depleted but not normal	Selgrade, et al., 1981
6,600	3.5	Continuous or Intermittent (7 hr/day, 7 days/wk)	Mouse	Increased susceptibility to S. pyogenes aerosol with increased duration of exposure. No significant difference between modes of exposure	Gardner, et al., 1979
7,500-1,3000	4-7	Continuous 14 days	Mouse	Increase of injected radio-labeled protein in lung	Sherwin and Richters, 1971
8,100	4.5	1, 3.5 or 7 hrs	Mouse	Increased susceptibility to S. pyogenes aerosol proportional to duration of exposure. No effect when bacterial challenge was delayed 18 hrs.	Gardner, et al., 1981
9,400	5	3 hr	Guinea Pig	Increase protein and lipid content of lavage fluid in vitamin C-depleted but not normal after 18 hrs	Selgrade, et al., 1981

TABLE 14-21. (continued)

Concentration of NO_2 $\mu g/m^3$	 ppm	Time of exposure	Species	Summary of effects	References
9,400-94,000	5-50	3 hr	Rabbit	No measurable effect on benzo(a)pyrene hydroxylase activity of tracheal mucosa	Palmer, et al., 1972
9,400	5	4 hr	Guinea Pig	Increase in respiratory rate and decrease in tidal volume	Murphy, et al., 1964
9,400	5	4 hr/day 5 days/wk 2 mo.	Guinea Pig	Increased lung tissue serum antibodies	Balchum, et al., 1965
9,400	5	7.5 hr/day 5 days/wk 5.5 mo.	Guinea Pig	No increase in airflow resistance	Murphy, et al., 1964
9,400	5	14-72 hr	Mouse	Increased lung protein by radio-label method	Csallany, 1975
9,400	5	3 days	Guinea Pig	50% mortality; histological evidence of edema; increased protein and lipid content of lavage fluid in vitamin C depleted but not normal	Selgrade, et al., 1981
9,400	5	Continuous 1 wk	Rat	Hyperplasia began by 3 wks	Rejthar and Rejthar, 1974
9,400	5	Continuous 2 mo.	Monkey	Increased susceptibility to *K. pneumoniae*	Henry, et al., 1970

TABLE 14-21. (continued)

Concentration of NO_2 µg/m³	Concentration of NO_2 ppm	Time of exposure	Species	Summary of effects	References
9,400-18,800	5-10	Continuous 90 days	Monkey	Infiltration of macrophages, lymphocytes and some polymorphonuclear leukocytes; hyperplasia of bronchiolar epithelium and Type II cells	Busey, et al., 1974
9,400-18,800	5-10	Continuous 90 days	Monkey	No significant hematological effects	Coate and Badger, 1974
9,400	5	Continuous 133 days	Monkey	Immunization with mouse-adapted influenza virus. Initial depression in serum neutralization titers with return to normal by 133 days. No change in hemagglutination inhibition titers or amnestic response	Matsumura, 1970
18,800 190	10 0.1	6 hr	Mouse	No chromosomal alterations in leukocytes or primary spermatocytes	Gooch, et al., 1977

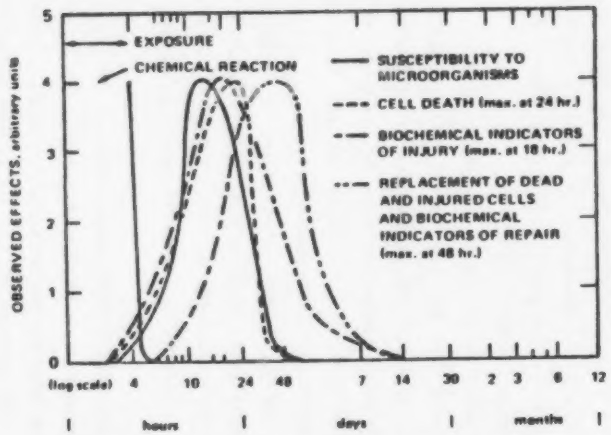


Figure 14-4. Temporal sequence of injury and repair hypothesized from short-term single exposures of less than 8 hours.

same for all mammalian species exposed under similar conditions. These reactions will be obtained predominately with low concentrations of NO_2. As the concentration of NO_2 is increased more than 100-fold over ambient concentrations, complications arise which tend to obscure the sequence of events. It is not clear, further, whether or not these higher concentrations are truly relevant to the toxicity of NO_2 to man as it occurs in the atmosphere of urban areas. In any event, the animal studies most important to determining the standards used in regulating emissions are those more closely aligned to ambient concentrations of NO_2.

Studies of the reaction of NO_2 with cellular constituents clearly illustrate that the chemical reactions are essentially instantaneous (Roehm et al., 1971; Menzel, 1976) when compared with the length of time required for demonstration of a biological effect. Most investigators believe that the chemical reactions of NO_2 are dominantly with lipid components of the cell (Menzel, 1976). The reaction of NO_2 with the unsaturated lipids of cellular membranes results in a chemical reaction characterized by the formation of peroxidic products. This is a devastating event in terms of the organization and properties of the cellular membrane necessary to maintain the integrity of the cell. Many of the biological effects can be ascribed to the peroxidation of cellular membranes, the most obvious example of which is pulmonary edema, a commonly observed phenomenon on exposure to high concentrations of NO_2. The protective role of vitamin E in the prevention of NO_2 toxicity at high concentrations is also supportive of this hypothesis (Menzel, 1976).

Alternatively, NO_2 could oxidize a number of small molecular weight compounds such as glutathione, pyridine nucleotides and ascorbic acid. Thiol oxidation could be coupled to lipid peroxidation through the glutathione peroxidase-glutathione reductase cycle (Chow et al., 1974). The increased pulmonary edema found in guinea pigs mildly depleted of vitamin C (ascorbic acid) suggests that vitamin C also plays an important role in the maintenance of cellular integrity during NO_2 exposures (Selgrade et al., 1981).

Inhaled NO_2 is rapidly taken up and distributed throughout the lung as has been determined using short lived radiotracer studies with $^{13}NO_2$ (Goldstein et al., 1977b). A very significant fraction of NO_2 is retained in the lung. The fraction retained probably represents that NO_2 which is chemically reactive with pulmonary tissue via addition to unsaturated fatty acids.

NO_2 is an acid anhydride and reacts with water vapor at ambient concentrations in the air and more so at the increased temperature and humidity existing within the respiratory system. The exact chemical species which reaches the pulmonary surface to produce the observed lesions is most likely NO_2, but HNO_2, and perhaps NO may be formed in the liquid lining the airways (Goldstein et al., 1977b). HNO_2 and HNO_3 will be rapidly neutralized by the biological substances dissolved in the liquid layer lining the airways of the lung.

Despite the hydration of NO_2 by water vapor, a significant fraction of NO_2 is not removed in the upper airways and penetrates deep within the lung to produce its toxic effects. As a strong oxidant, NO_2 may also oxidize small molecular weight reducing substances and proteins within seconds to minutes. Reaction with unsaturated fatty acids to produce peroxidation is essentially instantaneous (Roehm et al., 1971). It is not likely, so far as is known, that NO_2 reaching the respiratory portions of the lung would be able to penetrate the lung cells and attain a significant concentration within the blood. Nitrate is formed as a consequence of reactions with cellular constituents and has been detected in the blood and urine of animals exposed to NO_2 (Kosmider, 1975). Levels of nitrate attained during NO_2 exposure are unlikely to induce biological responses of the nature which have been observed. Because of the high reactivity of NO_2, the predominant response observed on the inhalation of NO_2 is direct injury to the tissues of the lung. The effects on organs distal to the lung are likely to result from the production of secondary toxicants in the lung which are circulated to other parts of the body. A direct proof of this hypothesis of circulating toxins following inhalation of NO_2 has not been found, but effects on organs other than the lung have been found (Miller et al., 1980). The metabolism of xenobiotic compounds, for example pentobarbital, by the liver is inhibited by exposure of mice to 470 $\mu g/m^3$ (0.25 ppm) NO_2 for 3 hours (Miller et al., 1980). Repeated exposure to NO_2 resulted in a return to normal metabolism of pentobarbital. While the implications of these observations are difficult to understand, decreased drug metabolism represents the inhibition of an important detoxification pathway in the liver for a variety of compounds to which man is exposed both intentionally (drugs, pesticides, and food additives) and unintentionally (naturally occurring or adventitious toxicants, mutagens, and carcinogens). Inhibition of drug metabolism is generally recognized as an adverse effect of a drug, and thus, these effects are also adverse. Future studies on the effects of NO_2 inhalation on other organ systems deserve continued surveillance.

The major pulmonary effect of NO_2 is cellular injury among specific cell types within the lung (Freeman et al., 1968b). If the NO_2 injury is severe, cell death results. These events occur within 24 hours after inhalation. The magnitude and site of the injury resulting from NO_2 will depend upon the concentration of NO_2 which was inhaled; therefore, the absolute degree of response will depend upon both the rate and magnitude of respiration and the NO_2 concentration. The moderate solubility of NO_2 in water and the inability of the upper respiratory tract to remove all of the NO_2 which is inhaled result in injury to specific regions within the lung. At concentrations near those found in urban environments, the region of the lung bounded by the terminal and respiratory bronchioles and adjacent alveoli are those which are most affected (Freeman et al., 1968,b,c; 1969b, 1974a). Emphysematous alterations have been reported in mice exposed for 6 months to 188 $\mu g/m^3$ (0.1 ppm) NO_2 with a daily 2-hour spike of 1,880 $\mu g/m^3$ (1 ppm) NO_2 (Donovan et al., 1976). The bronchiolar region represents the terminal portion of the lung and is intimately involved in the exchange of oxygen and carbon dioxide. This is the region of the lung which is most essential for the maintenance of life. Some differences may exist between man and rodents because this region of the lung is proportionately much shorter in the rat than in man. At high concentrations of NO_2, that is above ~9,400 μg/m^3 (~5 ppm), segments of the upper airways, as well as those centering around the alveoli, may be affected. As cells are exposed to NO_2 and begin to die, protein and nucleic acid synthesis is stimulated in the surviving stem cells and a wave of mitosis occurs which reaches its maximum at about 48 hours during or after exposure. The type I cell of the lung (a thin, squamous cell across which gases are exchanged) appears to be the most sensitive and to be injured at lower concentrations than the type II cell (a cuboidal cell that produces surfactant) (Evans et al., 1973b). The nature of this injury can be sufficiently severe that the cell dies, sloughs off, and leaves debris within the alveoli. Other cells in the upper airway, such as ciliated cells, are similarly sensitive and may be replaced by other stem cells known as Clara cells. These effects on the lung result in dramatic changes in its structure and cell composition.

Swollen collagen fibers occur in rabbits exposed to 470 μg/m^3 (0.25 ppm) NO_2 for 4 hr/day, 5 days/wk for 24 days (Buell, 1970). Presumably, these swollen collagen fibers represent alterations in the basement membrane or disruptions in collagen synthesis. One can not speculate on the potential pathological sequelae of collagen dysfunction, except to note that collagen metabolism is disrupted in man and animals during fibrosis. Some alterations in collagen metabolism are suggested by the long-term exposures of dogs to auto exhaust containing NO and NO_2 (Orthoefer et al., 1976). Increased number and size of interalveolar pores found in these dogs after near life-time exposures (Hyde et al., 1978) emphasize the importance of the emphasematous alterations found in mice on exposure to NO_2 alone (Donovan et al., 1976).

Biochemical indicators of lung injury can provide early evidence of toxicity. The rat lung prostaglandin dehydrogenase is sensitive to exposures of as little as 3 hr to 376 μg/m^3 (0.2 ppm) NO_2 (Menzel, 1980). In this series of experiments, rats were exposed to 376, 3,760 or 35,720 μg/m^3 (0.2, 2 or 19 ppm) for 3 hours. The lungs were then removed and used as an isolated, ventilated and perfused lung preparation to determine the uptake, release and metabolism of prostaglandin E_2 (PGE_2), a natural vasoactive hormone secreted by the lung and other organs. NO_2 exposure inhibited the metabolism of PGE_2 to its inactive metabolite 13,14-dehydro-15-keto PGE_2. While there was no difference in the amount of inhibition produced by exposure to 376 and 3,760 μg/m^3 (0.2 and 2 ppm) NO_2, the time required to return to basal levels of activity was greater for those rats exposed to 3,760 μg/m^3 (2 ppm) than to 376 μg/m^3 (0.2 ppm). Rats exposed to 35,720 μg/m^3 (19 ppm) NO_2 required the longest time for recovery to basal levels. Recovery required 60 hours following exposure to 376 μg/m^3 (0.2 ppm), 90 hours following 3,760 μg/m^3 (2 ppm) and 160 hours following 35,720 μg/m^3 (19 ppm). No alteration of the uptake or release of either ^3H-PGE_2 or its metabolites by the exposed rat lungs was found. None of the lungs examined showed any evidence of edema as judged by the wet weight to dry weight ratio. Since the prostaglandin system is intimately involved in the local regulation of blood flow in the lung, alterations in the metabolism of these potent hormones may have profound effects on the perfusion of the lung and subsequently on the gas exchange of the affected lung.

As pointed out above, vitamin C appears to play an important role in the maintenance of the integrity of the airways to macromolecules. Selgrade et al. (1981) repeated in part the experiments of Sherwin and Carlson (1973) in which guinea pigs were depleted of vitamin C and exposed to NO_2. Selgrade et al. failed to find an increase in the lavage fluid protein or lipid content of guinea pigs exposed to 740 $\mu g/m^3$ (0.4 ppm) NO_2, even after exposure for 2 weeks. Exposure to 1,880, 5,640 or 9,400 $\mu g/m^3$ (1, 3 or 5 ppm) NO_2 for 3 days caused increased protein and lipid content of the lavage fluid. A single exposure to 9,400 $\mu g/m^3$ (5 ppm) for 3 hours also produced similar changes in the lavage fluid content after 18 hours in vitamin C-depleted guinea pigs, but not in normal guinea pigs. Vitamin C depletion also caused a 50 percent mortality in the animals exposed to 9,400 $\mu g/m^3$ (5 ppm) NO_2, but no mortality in animals having normal vitamin C levels.

The differences between the Sherwin and Carlson and the Selgrade et al. studies may be explained by differences in the exposure systems, in the vitamin C status of the guinea pigs, and in the methods of analysis. Sherwin and Carlson exposed guinea pigs to NO_2 by the use of a silicon drip technique and monitored the NO_2 concentrations with a Mast Meter and intermittent Saltzman determinations.

Considerable improvement has been made in the methods of analysis for NO_2 and exposure of animals. Selgrade et al. used a chemiluminescent meter and a Hinners chamber for exposure. Considerable variation could have occurred in the chamber NO_2 concentration using the older methods. The degree of vitamin C depletion used by Selgrade et al. was quite mild, representing only a 25 percent reduction of the serum levels of the vitamin. No details are provided in the Sherwin and Carlson study to allow an assessment of the degree of vitamin C deficiency of the animals. More completely depleted guinea pigs could have been more susceptible. Sherwin and Carlson also used disc electrophoresis to estimate the protein concentration of the lavage fluid, while Selgrade et al. used the direct chemical method of Lowry et al. Additionally, Selgrade et al. found another protein not detected by Sherwin and Carlson. The new protein was detectable only on careful photometric scanning of the gels and could have gone undetected in the earlier study since considerable improvement in technique has been possible by advances in the field of protein chemistry, especially electrophoresis.

These studies illustrate that vitamin C deficiency may lead to greater susceptibility to ambient levels of NO_2. Burch (1961) compared the vitamin C levels reported in guinea pigs and in human autopsy tissue samples from a number of organs. Guinea pig tissue ascorbate concentrations reach saturation levels on continued intake of ascorbate. Since human leukocytes also reach saturation levels on high vitamin C intake, a similar saturation curve for human tissues is likely. Thus, the 25 percent reduction in serum vitamin C levels that resulted in these major changes in susceptibility to NO_2 represent conditions likely to occur in man with low vitamin C intake. Burch found 29 percent of 1,000 subjects in 8 high schools to have serum ascorbate levels below 0.4 percent, an arbitrary biochemical deficiency level, while 48 percent of 150 students from a poor area had values below 0.4 percent. Oral contraceptive use (Wynn, 1975) and smoking (Hoefel, 1977) also lower the blood ascorbate levels in man. At present the dose response curve for vitamin C intake and changes in lung permeability by NO_2 exposure are not known, so one cannot relate the changes reported by Selgrade et al. quantitatively to the present levels of vitamin C intake by the U.S. population and the risk of persons having low vitamin C to adverse effects associated with NO_2 exposure. The subject is obviously important and should be reviewed as further information becomes available.

Another sensitive measure of injury is the appearance of proteins in the airways. Increased protein content of the pulmonary lavage fluid or the appearance of radiolabeled serum proteins in the airways is taken as an indicator of pulmonary edema. Radiolabeled albumin or rabbit serum proteins injected into the blood stream of mice were detected in the airways after exposure to 7,520-13,170 $\mu g/m^3$ (4-7 ppm) NO_2 (Sherwin and Richters, 1971). Under normal circumstances, such exudates of serum proteins do not occur and represent a major, deleterious alteration in the permeability of the airways. While there was a trend toward increased amounts of radiolabeled protein in the airways of exposed mice, the results were not statistically supportable, mainly because of the large variation in the values. These variations could have been due to variations of the NO_2 concentration during exposure since the exposure ranged from 7,520 to 13,170 $\mu g/m^3$ (4-7 ppm) or to variable recovery of the radiolabeled protein in tissue extracts. Since the time of exposure to discrete levels of NO_2 is not known and since only trends toward increased levels of proteins within the airways were reported, one must conclude that exposure to 7,520-13,170 $\mu g/m^3$ (4-7 ppm) NO_2 does not produce pulmonary edema in normal mice.

The effect of NO_2 inhalation on the metabolism of the carcinogen benzo(a)pyrene by the lung has been investigated. Palmer et al. (1972) found no effect on benzo(a)pyrene metabolism of the tracheobronchial region of rabbits exposed to 9,400 $\mu g/m^3$ (5 ppm) NO_2 and greater. Law et al. (1975) found no effect of short-term exposures at even higher levels (75,200 $\mu g/m^3$; 40 ppm for 2 hours) on rat lung microsomal benzo(a)pyrene metabolism. NO_2 exposure appears to have no effect on these cytochrome P_{450}-dependent enzyme systems.

There is no reason to suppose that all enzymes are equally sensitive to NO_2 exposure, so lack of an effect on some enzymes is not indicative of an absence of a toxic effect at that NO_2 concentration. Further, some enzymatic changes, such as increased acid phosphatase levels (Sherwin and Carlson, 1973), may represent changes in the cell population due to death of some cells and replacement by a new younger cell population.

Pulmonary defenses against infectious agents are affected by short-term exposures to NO_2. The infectivity model in which pollutant-exposed animals receive an aerosol of live microbes has proven to be a particularly sensitive indicator of pulmonary injury and has been responsible for the development of most of the data indicating toxicity of NO_2 at low concentrations and short times of exposure (Coffin et al., 1976; Gardner and Graham, 1976; Ehrlich, 1975).

Mortality from exogenous infectious agents is influenced more in proportion to the concentration of NO_2 than to the duration of exposure. This observation is consistent with the hypothetical temporal sequence of injury. Pulmonary damage occurs rapidly on exposure to NO_2 but the functional effects of pulmonary damage may be observed much later, depending upon the extent of damage and the system which has been used to measure the damage; e.g., pulmonary conversion of PGE_2 to its metabolites (Menzel, 1980) or susceptibility to airborne infections. (Coffin et al., 1976; Gardner and Graham, 1976; Ehrlich, 1975) The infectivity model tends to be an integral of many of the defensive mechanisms of the lung and, therefore, to reflect the overall damage which has occurred. NO_2 concentrations as low as 4,700 μg/m^3 (2.5 ppm) may result in excess mortality from a single exposure of only 3 hours (Ehrlich et al., 1977). The injury of a 3-hour exposure appears to be repaired within 24 to 36 hours after exposure, but not by 18 hours (Selgrade et al., 1981).

In Figure 14-5 a short-term exposure of constant duration has been given to an animal and only one of the properties of intoxication is illustrated, the death of type I cells. Increasing concentrations of NO_2 are illustrated in this figure on the z axis. Thus, as the concentration of NO_2 is increased, the magnitude of cellular death increases while the time at which cell death occurs is constant. The magnitude of cell death is proportional to the logarithm of the concentration of NO_2 which has been inhaled. Increasing the total amount of NO_2 inhaled by manipulations in the respiratory pattern will likewise increase the magnitude of cell death, but not influence the time at which cellular death occurs. Mice made to run and to inhale more and more deeply NO_2 are more susceptible to airborne infection than those resting (Selgrade et al., 1981). Eventually, sufficient cells will be injured to produce mortality during the peak wave of death of alveolar cells. Death through respiratory insufficiency and pulmonary edema, however, does not occur at concentrations achievable in the urban atmosphere. Concentrations greater than 47,000 μg/m^3 (25 ppm) are necessary to achieve mortality. This is not to say, however, that severe pulmonary damage is not achieved at lower concentrations near those which occur regularly in urban areas.

The delay between end of exposure and observation of biological effect complicates the understanding of the effects of long-term exposure to NO_2. This is especially so with exposure regimens resembling those that occur in the atmosphere where exposure to relatively high concentrations of NO_2 may occur repeatedly over a short time period. Figure 14-6 illustrates the sequence of events which is hypothesized to occur on continuous long-term exposure to NO_2. The sequence of events is essentially similar to that in short-term exposure. The chemical reactions between the inhaled NO_2 and cellular constituents are instantaneous and achieve a constant level throughout the exposure. During the first 14 days of exposure, cell death and replacement of pulmonary cells are the dominant features. This is expressed as a wave of mitosis or cellular division which reaches its maximum about 48 hours after the onset of exposure. The extent of cell death is illustrated in Figure 14-6 and is proportional to the concentration of NO_2. Likewise, all of the other indicators of NO_2 damage so far examined are

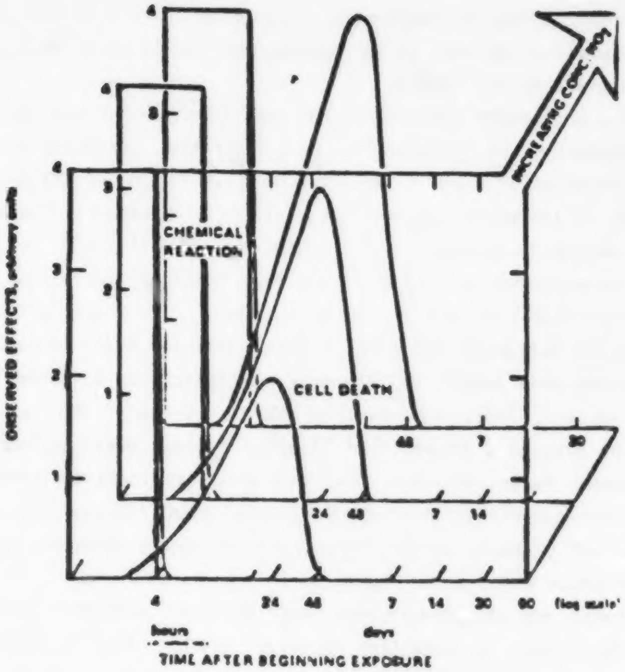


Figure 14-5. Proportionality between effect (cell death) and concentration of NO_2 during a constant exposure period. The maximum in cell death is reached ~ 18 hours after exposure and the extent is proportional to the dose (concentration x time).

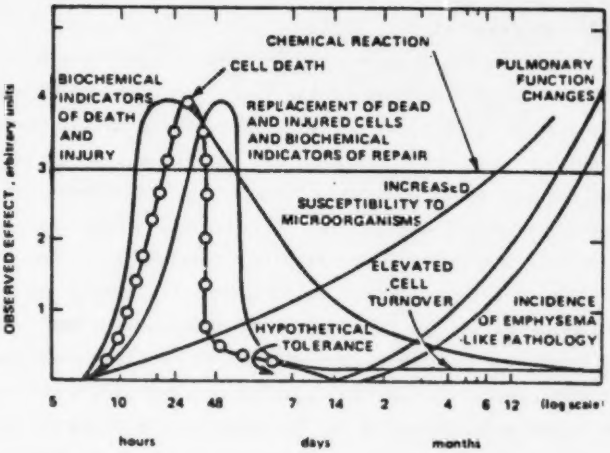


Figure 14-6. Temporal sequence of injury and repair hypothesized from continuous exposure to NO_2 as observed in experimental animals.

dose dependent. The biochemical and physiological functional indicators of damage change rapidly with injury and repair, reaching a relatively steady state after about a week or two (Menzel et al., 1972; Donovan et al., 1976; Menzel et al., 1977). Several enzymes have been detected which are indicative of cellular injury at concentrations of NO_2 as low as 376 $\mu g/m^3$ (0.2 ppm) (Menzel, 1980) or 940 $\mu g/m^3$ (0.5 ppm) (Donovan et al., 1976; Menzel et al., 1977) during the injurious phase of continuous exposure, that is greater than 7 days. Pulmonary macrophages are aggregated within the lung and the degree of aggregation has been estimated by a number of biochemical techniques (Aranyi et al., 1976; Ehrlich et al., 1975). Again, the infectivity model is highly sensitive to NO_2 exposure. The susceptibility to infection as measured by this technique rises almost linearly during this period. The infectivity model has been used to illustrate excess mortalities due to NO_2 exposure at concentrations within the range of 940-2,820 $\mu g/m^3$ (0.5-1.5 ppm) NO_2 (Gardner et al., 1979; Ehrlich and Henry, 1968; Freeman et al., 1972; Stephens et al., 1971).

Long-term exposures to NO_2 also result in major alterations of lung morphology. These are very difficult to interpret because of the fine gradation and slow development of response once the initial phase of replacement of cells susceptible to NO_2 has passed (Freeman et al., 1968c, 1972; Stephens et al., 1971; 1972). The development of an emphysema-like disease in experimental animals requires considerable time as has been demonstrated in studies of rats and mice. The development of obstruction to airflow, distension and destruction of the alveolar tissue in experimental animals requires considerable time (Freeman et al., 1972). When compared to the life span, the time required for damage in experimental animals is equivalent to that required for the development of emphysema in man. The process of emphysema development on NO_2 exposure is indeed complex, but it is clear that the effects are interpretable in terms of the changes in the cell populations and structural alterations concommitant to that. A major pathologic change is an increase in the distance between the air space and the capillary in the respirable or alveolar region of the lung (Henry et al., 1970; Evans et al., 1972, 1974; Buell, 1970). Other effects include the loss of ciliated cells which are responsible for removing particles from the lung and narrowing of the airways and alteration in the morphology of the cells lining the junction between the respiratory segment and the mucous containing segment (Stephens et al., 1971, 1972). The cell type in the alveoli most sensitive to NO_2, the type I cells, is replaced by type II cells, the type I cell progenitor, but the appearance of the type I cells maturing in the presence of NO_2 is significantly different from those maturing in the absence of NO_2 (Evans et al., 1974). Other alterations in the lung include the appearance of collagen in areas which are normally devoid of this fibrous protein and the aggregation of macrophages (Stephens et al., 1971; Buell, 1970). These effects have been observed in rats that have been exposed continuously to 3,760 $\mu g/m^3$ (2 ppm) NO_2 or greater.

The fatty acid composition of the lung membranes has also been noted to change during the exposure to NO_2 (Menzel et al., 1972). The mortality from continuous exposure to high concentrations of NO_2 is influenced by the level of vitamin E and other free radical scavengers which are included in the diet (Menzel, et al., 1972). These observations support the hypothesis that membrane damage by chemical oxidation of unsaturated fatty acids is a major mechanism of toxicity of NO_2. These changes in fatty acid composition of the lung are accompanied by enzyme changes which in part may be protective and aid in the destruction of the peroxidic products formed in the lung on NO_2 inhalation (Chow et al., 1974; Donovan et al., 1976). It should be emphasized that at no point is it possible to provide adequate levels of vitamin E or other dietary factors that provide complete protection against NO_2. It should be noted, however, that certain segments of the population may be unusually sensitive to NO_2 should their intake of vitamin E and other antioxidants be marginal. A similar conclusion can be reached with regard to vitamin C intake. Normal levels of vitamin C provide protection when compared to marginal or deficient levels, but still do not prevent completely NO_2 toxicity. The level of vitamin C intake in some populations may also be marginal as discussed above.

The temporal sequence of events suggests that the response of the animal to inhalation of NO_2 returns to near normal levels during continuous exposure. These results are misleading, since it has clearly been observed in long-term studies of rats that the morphology of the lung has changed from its normal structure to that resembling emphysema (Freeman et al., 1968c; 1972; Stephens et al, 1972). This raises the question of tolerance to resistance and recovery during continuous exposure. It is possible that, at some point, the rate of replacement of dead and injured cells resulting from the continuous inhalation of NO_2 may return to levels equivalent to that found in clean air (Stephens et al., 1972). Tolerant cells, as compared to naive cells, may be more resistant to NO_2 because they are younger or because they may have produced a protective mechanism such as specific increases in enzymes capable of degrading the secondary reaction products formed on NO_2 inhalation. Some enzymes such as glutathione peroxidase, glutathione reductase, and glucose-6-phospate dehydrogenase in the rat may be increased as a protective mechanism (Chow et al., 1974), or they more likely may reflect a proliferation of young cells within the organ which contain higher enzyme concentrations (Sherwin et al., 1974). These cells are younger because all of the lung cells are dying and being replaced at a more rapid rate than that which would occur normally in the lung in pollutant free air. Not all species exhibit this protective increase in enzymes. For example, the guinea pig, when exposed to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 4 months failed to develop higher levels of these potentially protective enzymes (Donovan et al., 1976; Menzel et al., 1977). Importantly, when cultured lung cells are coated with a very thin layer of nutrients to resemble the condition within the lung, direct exposure to NO_2 is highly toxic. It is most likely that all cells are sensitive to relatively low concentrations of NO_2 and that no adaptation in the true sense ever occurs. The apparent adaptation that may be seen in pulmonary function measurements of people living in polluted areas vs. those who live in nonpolluted areas may be artifactual in the sense that large changes in pulmonary tissue may be necessary before permanent alterations may be detected in pulmonary function. In other words, a major pathophysiologic change must occur before it is detected by pulmonary physiology methods. Biochemical and morphological techniques are more sensitive, but so invasive that they can only be used on experimental animals. In further support of the idea that all cells are sensitive to concentrations of NO_2 which are easily attainable in inhaled air, when rats have been exposed to 3,760 $\mu g/m^3$ (2 ppm) NO_2 for long periods of time and then are exposed to an abrupt increase in concentration of NO_2, a second wave of mitosis and subsequent alterations in biochemical, physiologic, and morphologic indicators of cell damage occur in exactly the same temporal sequence (Evans et al., 1972). Thus, although "adaptation" may appear to occur, the ultimate development of an emphysema-like condition occurs in the rats on long-term exposure, and they remain sensitive to alterations to higher concentrations of NO_2.

An important consideration has been the question of tumor formation or malignant metaplasia due to NO_2 exposure. This concern comes about due to the morphology of the lungs of animals which have been exposed to NO_2. Because NO_2 produces a stimulation or rapid turnover of cells, a transient hyperplasia of the type II lung cell and nonciliated bronchiolar cell is observed. Such a hyperplasia represents a part of the natural repair mechanism. There is, however, no evidence to indicate that such changes represent tumor formation or malignant metaplasia. Thus, there is no data to connect the inhalation of NO_2 with an increased incidence of cancer at the present time.

As was noted in the discussion of the effects of short-term exposure to NO_2, the lag between exposure and biological effect represents a potential situation for accumulation of biological effects. Such a cumulative effect has been demonstrated using the infectivity model in mice which were exposed continuously or intermittently (7 hr/day) to 2,820 $\mu g/m^3$ (1.5 ppm) NO_2 (Gardner et al., 1979). Intermittent exposures of mice to 2,820 $\mu g/m^3$ (1.5 ppm) NO_2 eventually become equivalent to continuous exposure when the infectivity model is used. A total of 319 hours of exposure (13.3 days) is required before a 7 hour/day exposure becomes equivalent to continuous exposure. The time period for equivalence between intermittent and continuous exposures will be shortened relative to the concentration of NO_2. The intervening 17 hours between each 7 hour exposure to 2,820 $\mu g/m^3$ (1.5 ppm) NO_2 are inadequate for complete recovery. Excess mortality upon challenge with bacterial pathogens could be observed after 7 days of continuous exposure to 2,820 $\mu g/m^3$ (1.5 ppm), however, or even to 940 $\mu g/m^3$ (0.5 ppm).

Considerable differences occur in the response to NO_2 when animal species and infectious agents other than mice and S. pyogenes are used (Purvis and Ehrlich, 1963; Henry et al., 1970; Fenters et al., 1973; Matsumura, 1970a). Resistance to K. pneumoniae (Purvis and Ehrlich, 1963) is less affected by NO_2 exposure than resistance to S. pyogenes. Squirrel monkeys (Henry et al., 1970) were the least sensitive animals tested using this end point, and hamsters (Ehrlich, 1975) demonstrated intermediate sensitivity when compared to mice. The quantitative extension of such data to man is difficult because analogous human data are not available. Even though both infectious agents are human pathogens, the direct extension of this data to man is difficult, in part, because of differences in anatomical structure of the lung, and in part, because of differences in native and acquired immunity.

The recent work of Gardner et al. (1981) on the effects of short-term exposures to spikes of NO_2 similar to those occurring in the urban atmosphere is particularly important in assessing the interaction of NO_2 and pulmonary infections. Mice were exposed to spikes of 8,100 $\mu g/m^3$ (4.5 ppm) NO_2 for 1, 3.5, or 7 hours, and increased mortality due to exposure to S. pyogenes was proportional to the duration of the exposure. The mice recovered from the exposure by 18 hours. To mimic the urban environment, these same spikes were superimposed on a background of 2,800 $\mu g/m^3$ (1.5 ppm) NO_2, which resulted in a significant ($p < 0.05$) increase in mortality with a spike of 3.5 or 7 hours duration, when the bacterial challenge was delayed for 18 hours after the peak NO_2 exposure. These results are consistent with the long-term studies of this group using the same model system where it was found that the magnitude of the exposure was more important than the product of time and concentration. These data suggest that the alterations in the lung leading to increased susceptibility to airborne infections occur with relatively brief exposures approaching those encountered in the urban environment. These experiments provide an experimental basis for the observations relating increased incidence of respiratory infections with gas stove exposure in man (References ERC/RTP Review, 1976; Melia et al., 1978; Spengler et al., 1979; Speizer et al., 1980; Goldstein et al., 1979 from Chapter 15). The effect of cooking stoves on respiratory infections is discussed more fully in Chapter 15 (Section 15.2.2.2.2). Levels of 470 to 1,100 $\mu g/m^3$ (0.25 to 0.6 ppm) NO_2 were found in the vicinity of gas stoves for about 2 hours (References ERC/RTP Review, 1976; Mitchell et al., 1974 from Chapter 15).

In terms of the probable temporal sequence of events, NO_2 inhalation affects almost all of the cell types within the lung. Depending, then, upon the concentration of NO_2, different cells will be affected in addition to those which are most susceptible at lower concentrations. The mode of exposure, that is the rate and depth of respiration, will also influence the specific cell types which are damaged. Because the rate of chemical reaction of NO_2 with cell constituents is almost instantaneous when compared to the time required for biological expression of injury, it may be expected that the concentration of NO_2 during a given exposure will have a greater effect on determining the end point used to measure toxicity than would the duration of exposure. Sensitive biochemical parameters are difficult to interpret because of the need to correlate biochemical changes with pathological processes. They are viewed, then, as indicators of death or injury of specific cells following NO_2 inhalation. Biochemical studies indicate that lung injury occurs on inhalation of NO_2 at levels as low as 376 $\mu g/m^3$ (0.2 ppm) for 3 hours in mice, as evidenced by changes in prostaglandin metabolism (Menzel, 1980); as 470 $\mu g/m^3$ (0.25 ppm) for 3 hr/day for 1 day as evidenced by increased pentobarbital metabolism in mice (Miller et al., 1980); as 940 $\mu g/m^3$ (0.5 ppm) for 8 hr/day for 7 days as evidenced by alterations in serum enzymes in guinea pigs (Donovan et al., 1976; Menzel et al., 1977); as 940 $\mu g/m^3$ (0.5 ppm) continuous exposure with 1-hour peaks of 3,760 $\mu g/m^3$ (2 ppm) for 5 days/wk in mice as evidenced by morphological alterations in alveolar macrophages, decreased serum antibody and immunoglobins (Ehrlich et al., 1975); as 1,000 $\mu g/m^3$

(0.53 ppm) for 8 hr/day for 180 days in guinea pigs as evidenced by alterations in serum enzymes (Drozdz et al., 1975); and as 1,880 $\mu g/m^3$ (1 ppm) continuously for 2 weeks in rabbits as evidenced by decreased lecithin synthesis (Seto et al., 1975). Not all effects have the same sensitivity; for example, guinea pigs exposed to 740 $\mu g/m^3$ (0.4 ppm) NO_2 have no alteration in lung permeability to serum proteins but do at 1,880 $\mu g/m^3$ (1 ppm) when rendered slightly deficient in vitamin C (Selgrade et al., 1981).

Because of the universal toxicity of NO_2 to pulmonary cells, it is likely that other air pollutants such as ozone, sulfuric acid, sulfur dioxide, and particulate matter may injure the same cells within the lung as are injured by NO_2. In most cases, following the simultaneous inhalation of NO_2 and other air pollutants, additive, rather than synergistic, effects have been found. Tobacco smoking and occupational exposure add very significantly to the toxicity of NO_2. At present, the data are not sufficient to provide a detailed evaluation of this important variable in the response of the human population. Because of the delay between the exposure to NO_2 and effect, the sequence of exposures to air pollutants may be particularly important. No synergism occurs between O_3 and NO_2 at the lowest concentrations examined for each pollutant of 100 $\mu g/m^3$ (0.05 ppm) with spikes of 200 $\mu g/m^3$ (0.1 ppm) (Gardner et al., 1981). At 940 $\mu g/m^3$ (0.5 ppm) NO_2 with 1,880 $\mu g/m^3$ (1 ppm) spikes combined with 0.05 ppm O_3 with 0.1 ppm spikes or higher, synergism between O_3 and NO_2 occurs using mice in the infectivity experiment (Gardner et al., 1981).

Another area of possible toxicity may be the formation of nitroso compounds, because nitrosamides and nitrosamines are known carcinogens. Nitrosamines and nitrosamides have recently come into the public view through their formation in foodstuffs containing nitrites. In this case, nitrite has been added to the foodstuffs to prevent bacterial contamination and spoilage. Gas phase reactions between NO_2 and amines to form nitrosamines have been reported, and inhaled, injected or ingested nitrosamines produce lung tumors in exposed animals. At the moment, no evidence exists that nitrosamines or nitrosamides are formed in ambient air from nitrogen oxides. The detection of nitrosamines in the body of mice gavaged morphroline and breathing NO_2 (Iqbal et al., 1980) suggests that nitrosamine formation in the lung can occur. The contribution of inhalation vs. ingestion as sources of nitrosamines remains to be quantified. Similarly, the role of inhaled nitrites and nitrates found in atmospheric particles is unknown and should be studied further. A few experiments indicate that inhaled nitrate produces biological effects through the release of histamine and other intracellular hormones. Whether such effects occur in man is not known. Continued surveillance of these important areas is needed.

While much remains to be learned about the toxicity of NO_2, studies so far conducted in animals indicate that the biological effects of NO_2 are likely to be displaced from the time of exposure. As shown in Figures 14-4 through 14-6, this delay between onset of symptoms and exposure to NO_2 may explain many of the confounding factors observed in epidemiologic data, but complicates further the question of effects of transient episodes of high NO_2 concentrations in the atmosphere. It is clear that the lowest concentration at which NO_2, in particular, produces biological effects of a reproducible magnitude so far detected in animals is 376 µg/m^3 (0.2 ppm). Repeated exposures to 940 µg/m^3 (0.5 ppm) also produce measurable adverse effects. The observation of cumulative effects is especially important, suggesting that under appropriate circumstances intermittent short-term exposure to NO_2 may eventually become equivalent to continuous long-term exposure. These observations, when compared to the data which have been accumulated on the long-term effects of NO_2, may be particularly pertinent to the potential toxicity of this air pollutant to man. There is little doubt that the inhalation of NO_2 results in toxicity, regardless of the species which has been exposed. Thus, animal experiments are truly indicative of the hazard of this air pollutant to man.

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15. EFFECTS ON HUMANS OF EXPOSURES TO OXIDES OF NITROGEN

15.1 INTRODUCTION

The present chapter discusses effects of oxides of nitrogen (NO_x) on human health, with major emphasis on the effects of nitrogen dioxide (NO_2) as the NO_x compound currently of greatest concern from a public health perspective. Human health effects associated with exposure to nitrogen dioxide (NO_2) have been the subject of three literature reviews since 1970. Each focussed mainly on the effects of short-term exposures.

In the first review, published in 1971, the Committee on Toxicity of the National Academy of Sciences-National Research Council issued guides to short-term exposures of the public to NO_x (National Academy of Sciences, 1970). A review of the then current literature showed: (1) that the individuals most susceptible to NO_2 action are those predisposed by age, heredity, and preexisting respiratory disease; (2) that many of these individuals respond most sensitively at concentrations to which healthy individuals are unresponsive; and (3) that the effects of NO_2 are, within limits, reversible such that the extent of recovery seems to be a function of the degree of exposure, the length of the interval between exposures, and the health and/or age of the exposed individuals. This committee acknowledged that too much critical information was missing to permit highly conclusive recommendations about short-term exposure to NO_2, but did conclude that the exposure limit for the general public for 10 minutes, 30 minutes, or 60 minutes should be established at 1,880 $\mu g/m^3$ (1.0 ppm).

A more complete review was published by the National Academy of Sciences in 1977 (National Academy of Sciences, 1977). This document covered the medical, biological, and physical effects of the nitrogen oxides and made recommendations for future research, but included no suggestions for legislative exposure limitations.

A task group on environmental health criteria for oxides of nitrogen, representing the World Health Organization, recommended in 1978 that the effects of pollution designated as "adverse", in addition to the morphological and other changes produced by high NO_2 concentrations, should include: increased airway resistance, increased sensitivity to bronchoconstrictors, and enhanced susceptibility to respiratory infections (World Health Organization, 1977). This group selected 940 $\mu g/m^3$ (0.5 ppm) as their estimate of the lowest concentration of NO_2 at which adverse health effects due to short-term exposure might be expected to occur, but did not state their rationale explicitly.

The following assessment appraises most of the studies included in these previous reviews plus additional, more recent, pertinent publications. This assessment includes discussion of controlled human exposure studies and epidemiological studies of human health effects associated with indoor and outdoor exposures to NO_x compounds. In addition, studies on the effects of accidental and occupational exposures to oxides of nitrogen are concisely reviewed in this chapter.

Placing the present assessment in historical perspective, it should be noted that concern regarding NO_x effects on human health was originally derived from cases of accidental or occupational exposures to NO_x compounds. For example, the earliest evidence for potential damage to man due to NO_x exposures occurred in the chemical industry where, as early as 1804, the deaths of a man and his dog after breathing nitric acid fumes were recorded (Raminez, 1974). Other occupational exposures have since been seen with the use of explosives which generate NO_2 during misfires and welding operations which generate substantial quantities of NO_x. Burning of plastics, shoe polish, and nitrocellulose also results in potentially excessive quantities of NO_x. High concentrations of ambient NO_2 were clearly associated with acute pulmonary edema and death. Lowry and Schuman (1956) were among the earliest investigators who demonstrated that exposures to NO_2 in excess of 200 ppm would induce such effects. Their description of silo fillers' disease clearly implicated NO_2 exposure in the etiology of this disease as another potential occupationally related NO_x hazard. That hazard, first noted in 1914 (Hayhurst and Scott, 1914), when four individuals died suddenly after entering a recently filled silo, was earlier mistakenly attributed to high concentrations of carbon dioxide.

A fairly typical clinical picture of signs and symptoms associated with exposures to very high levels of NO_x compounds (especially NO_2) has emerged from case studies of accidental and occupational exposures, as reviewed by Milne (1969) and Horvath et al. (1978). Acute exposure to high concentrations ($> 47{,}000\ \mu g/m^3$; 25 ppm) of NO_2 produces an almost immediate reaction consisting usually of cough, dyspnea, and tightness of the chest and respiratory tract caused by acute bronchitis or pulmonary edema. Such exposure may be quickly fatal. If the exposure has not been overly excessive, exposed individuals may recover without further complications. However, if the exposure concentration and duration are greater, more intense symptoms may occur after a latent period of 2 to 3 weeks. These consist of severe respiratory distress usually occurring quite suddenly and can result in death within a few days. The cause of these symptoms is always bronchiolitis fibrosa obliterans. When symptoms are not sufficiently severe to cause death, subjects frequently appear to recover fully. Such a biphasic reaction to exposure to NO_2 was described by Milne (1969), as determined from a literature review of clinical case studies.

Horvath et al. (1978) more recently reviewed data accumulated on 23 patients exposed to nitrogen dioxide in agriculture or industrial situations. Their review confirmed that, during the acute phase following severe NO_2 exposures, varying combinations of restrictive and obstructive ventilatory defects, impaired diffusion capacity and hypoxemia are found. However, Horvath et al. (1978) also noted some evidence for pulmonary dysfunction persisting after follow-up periods of 2.5 to 13.5 years, including diminished exercise tolerances with dyspnea during exertion. Fleming et al. (1979) recently conducted physiologic studies on a patient exposed to nitric acid fumes during the acute (immediate) and delayed (13 weeks later) stages. The acute changes were as anticipated. During the delayed stage, elastic recoil and resistance to flow were normal. However, dynamic compliance was reduced and dependent upon respiratory frequency and, more significantly, oxygen transport was abnormal during exercise with $P(A-a)O_2$ of 20 mm Hg. Dysfunction of small airways was evident. Many of the individuals who were discussed in the above case studies detected a pungent odor when initially exposed to NO_x compounds in various occupational situations.

The above types of effects noted in relation to accidental and occupational exposures (as discussed in more detail later in Section 15.4) helped to direct attention to possible human health effects induced by lower concentrations of NO_2 and other NO_x compounds encountered with indoor or outdoor exposures to ambient air pollutants. Further, the observations made on subjects inadvertantly exposed to clearly toxic levels of NO_x compounds contributed to the conceptual framework regarding possible functional changes associated with lower level NO_x exposures, the physiological bases of such changes, and the methodological approaches employed in attempting to measure such changes in animal toxicology, controlled human exposure, and epidemiological studies.

15.2 CONTROLLED HUMAN EXPOSURE STUDIES

Controlled exposure studies are useful because they can provide accurate measurements of the effects of specific exposures to single or simple combinations of pollutants. However, controlled studies usually do not provide definitive evidence of the effects that might be expected in ambient situations. In the uncontrolled natural environment, exposures are changing constantly in regard to both the mixture of pollutants present and the concentrations of each. The inability to extend controlled exposure studies over several months limits the extent to which human studies have been useful in determining the effects of repeated, short-term exposures to the highest levels of pollutants occurring in the natural environment.

For these reasons, controlled exposure studies usually are designed to determine the effects of a short, single exposure to a pollutant at a concentration believed to be high enough to produce some response. Obviously, in such studies the safety of test subjects is the overriding factor in determining the concentrations and exposure times that can be used. Initial estimates of these factors are often first obtained from animal studies. Prudence requires, however, that exposure times be limited to those causing initial responses. When effects are detected, additional studies are undertaken to collect evidence of the lowest concentration at which effects can be measured in both healthy and selected, sensitive volunteers. As noted above, the methods and physiological basis for most controlled studies are based on observations made on subjects inadvertently exposed to toxic levels of NO_2. These include controlled human exposure studies on the sensory effects of NO_x compounds as well as NO_x-induced pulmonary function changes discussed below.

15.2.1 Studies of Sensory Effects

15.2.1.1 Effects of Nitrogen Dioxide on Sensory Systems--The significance of effects on sensory receptors, if any, is unknown. The stimulation of a sensory receptor does invoke within an individual a specific response resulting from the neural transmission of the stimulation.

Changes in the intensity of light to which an individual is exposed initiate such a biochemically transmitted response. An impairment of dark adaptation, then, represents a slowing of the mechanism by which the eye adjusts to changes in light intensity. Because the response to the stimulus is delayed, this effect on dark adaptation must be considered to be an impairment. Whether or not it is reversible or whether or not the reversibility persists after each repeated insult, impairment of dark adaptation is reported at lower concentrations of NO_2 than is any other physiologic system tested.

Shalamberidze (1967) reported that impairment of dark adaptation occurred at NO_2 concentrations as low as 140 $\mu g/m^3$ (0.07 ppm) (Table 15-1). The tests, as reported by the investigator, demonstrated changes in ocular sensitivity to light by determining reflex changes in the functional state of the cerebral cortex. This study, however, gave results that conflicted with those reported by Bondareva (1963) (Table 15-1). This latter investigator determined normal dark-adaptation curves for five volunteers and then exposed them to concentrations of NO_2 varying from 150 to 500 $\mu g/m^3$ (0.08 to 0.26 ppm) to determine alterations that might be related to the changed atmosphere. While her results indicated that exposure to concentrations of 300 $\mu g/m^3$ (0.16 ppm) had no effect on dark adaptation, time for adaptation increased significantly as a result of exposure to 500 $\mu g/m^3$ (0.26 ppm). Repeated daily exposures to 500 $\mu g/m^3$ (0.26 ppm) NO_2 over a period of 3 months, however, induced an apparent physiological adjustment that largely reversed the initial increase in the time for adaptation. It has been suggested (National Academy of Sciences, 1977) that, since Bondareva did not indicate whether NO_2 was used alone or in combination with nitric oxide (NO), the higher value reported to be the minimum effective concentration may have been due to differences in the test atmospheres.

The perception of odors is a response to chemical stimulation of the olfactory receptors, but this perception fades with adaptation. The olfactory epithelium contains neural tissue which directly contacts airborne substances. Thus, interference with normal olfactory function could possibly reflect either direct or indirect interruption of neurochemical processes. Olfactory insensitivity to NO_2 could reduce awareness of potentially hazardous situations arising from increasing levels of the pollutant.

Studies of odor perception show that sensitive individuals can detect the characteristic pungent odor of NO_2 at a concentration of 200 $\mu g/m^3$ (0.11 ppm) (Table 15-1). Other studies have indicated that, by increasing relative humidity in the exposure atmosphere, odor perception is improved and respiratory irritation also is increased. Increasing the concentration gradually in the controlled exposure atmosphere results in a raising of the threshold so that its odor becomes less irritating.

Henschler et al. (1960) exposed groups of 20- to 35-year-old healthy males to NO_2 to obtain information on the lowest concentrations at which the odor would be detected immediately. The odor of NO_2 was perceived by three of nine volunteers when the concentration was 230 $\mu g/m^3$ (0.12 ppm), by 8 of 13 subjects when the concentration was 410 $\mu g/m^3$ (0.22 ppm), and by all of eight subjects when the concentration of NO_2 in the exposure chamber was 790 $\mu g/m^3$ (0.42 ppm).

TABLE 15-1. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON SENSORY RECEPTORS IN CONTROLLED HUMAN STUDIES

NO_2 Concentrations		No. of Subjects	Time until effect	Effects	No. of Subjects Responding	Reference
$\mu g/m^3$	ppm					
790	0.42	8	Immediate	Perception of odor of NO_2	8/8	Henschler et al., 1960
410	0.22	13	Immediate	Perception of odor of NO_2	8/13	Ibid.
230	0.12	9	Immediate	Perception of odor of NO_2	3/9	Ibid.
230	0.12	14	Immediate	Perception of odor of NO_2	most	Shalamberidze, 1967
200	0.11	28	Immediate	Perception of odor of NO_2	26/28	Feldman, 1974
0 to 51,000	0 to 27	6	54 minutes	No perception of odor of NO_2 when concentration was raised slowly from 0 to 51,000 $\mu g/m^3$	0/6	Henschler et al., 1960
2,260	1.2	6	Immediate	Perception of odor improved when relative humidity was increased from 55% to 78%	6/6	Ibid.
140	0.07	4	5 and 25 minutes	Impairment of dark adaptation	4/4	Shalamberidze, 1967
150 to 500	0.08 to 0.26	5	Initial	Increased time for dark adaptation at 500 $\mu g/m^3$ (0.26 ppm)	Not Reported	Bondareva, 1963
			Repeated over 3 months	Initial effect reversed		

The duration of exposure over which odor could be perceived varied considerably among subjects and was unrelated to the concentration of NO_2. At concentrations of up to 20,000 μg/m^3 (10.6 ppm), perception of the odor of NO_2 was lost after periods ranging from less than a minute to 13 minutes. However, regardless of the concentration, odor perception sensitivity returned within 1 to 1.5 minutes after subjects left the exposure chamber. Some subjects exposed to NO_2 concentrations as low as 230 μg/m^3 (0.12 ppm) reported a metallic taste, dryness, and constriction in the upper respiratory tract. Such symptoms lessened and eventually disappeared with repeated exposures. When NO_2 was added gradually to the exposure chamber over a period of approximately 1 hour, exposed subjects did not perceive the odor even when it reached a level of 51,000 μg/m^3 (27 ppm). When subjects were exposed to an atmosphere containing 2,260 μg/m^3 NO_2 (1.2 ppm) at 55 percent relative humidity, after which the humidity was increased very rapidly to 78 percent, a sharp increase in odor perception occurred along with a correspondingly sharp increase in irritation of the mucous membranes of the respiratory tract. It is possible that the increased irritation, and perhaps the increased odor perception, is the result of chemical reactions between nitrogen oxides and water in the controlled atmosphere that increase the concentrations of nitrogenous acids.

Odor perception studies were also conducted by Feldman (1974), who reported that 200 μg/m^3 (0.11 ppm) was the lowest concentration at which the odor of NO_2 was detected. At this concentration, 26 of 28 healthy subjects perceived the odor immediately.

In summary, controlled exposure studies indicate that two types of sensory receptors may be involved in the initial response in humans to the presence of NO_2. The sensory effects involved are the impairment of dark adaptation and the perception of odor. Effects were reported at levels as low as 140 to 200 μg/m^3 (0.08 to 0.1 ppm) and occurred almost immediately upon exposure.

15.2.1.2 Sensory Effects Due to Exposure to Combinations of Nitrogen Dioxide and Other Pollutants--Studies of the effects of NO_2 in combination with other pollutants on sensory receptors are summarized in Table 15-2. These studies, all from the Soviet Union, report that the effects of the test gases, when inhaled together, were additive, as they related to the minimum concentrations causing impairment of dark adaptation, odor perception, and changes in the amplitude of alpha rhythms in the brain.

Shalamberidze (1967) exposed 15 healthy subjects for periods of 5 or 25 minutes to various combinations of NO_2 and sulfur dioxide (SO_2). His studies were designed to compare the lowest concentrations at which dark adaptation is impaired by combinations of pollutants to levels of the individual gases causing impairment. He reported the minimum levels for impairment of dark adaptation for single pollutants to be 600 μg/m^3 (0.23 ppm) for SO_2 and 140 μg/m^3 (0.07 ppm) for NO_2. The thresholds for alteration of odor perception for subjects exposed to a single gas were 230 μg/m^3 (0.12 ppm) for NO_2 and 1,600 μg/m^3 (0.61 ppm) for SO_2. In these studies, combinations of the gases produced an impairment of dark adaptation or odor perception whenever the fractional threshold concentrations for the separate gases totaled one or more. The

TABLE 15-2. EFFECTS OF EXPOSURE TO COMBINATIONS OF POLLUTANTS ON SENSORY RECEPTORS IN CONTROLLED HUMAN STUDIES

Pollutant	Subjects	Exposure	Effects	Reference
Various combinations of NO_2 and other gases	15 healthy subjects	5 or 25 min; oral or nasal inhalation	The lowest effective concentration for dark adaptation was: NO_2, 140 μg/m^3 (0.07 ppm) and SO_2, 600 μg/m^3 (0.23 ppm). When inhaled together, the gases acted additively. Dark adaptation was impaired when the sum of the fractional threshold concentrations for the separate gases equaled 1.0 or more. Lowest effective concentration for odor perception was: NO_2, 230 μg/m^3 (0.12 ppm) and SO_2, 1,600 μg/m^3 (0.16 ppm). When inhaled together these gases acted additively. Odor was perceived when the sum of the fractional threshold concentrations equaled 1.0 or more.	Shalamberidze, 1967
Various mixtures of NO_x, SO_2, H_2SO_4 aerosol and NH_3	Not reported	Not reported	Lowest effective concentrations for odor perception of a combination of gases were reported to be: NO_x 20 μg/m^3 (0.01 ppm); SO_2 170 μg/m^3 (0.06 ppm); H_2SO_4 aerosol, 110 g/m^3 (0.03 ppm), and NH_3, 300 μg/m^3 (0.43 ppm). When inhaled together the odor was perceived whenever the fractional threshold totaled 1.0 or more.	Kornienko, 1972
Mixture of NO_x, SO_2, NH_3, H_2SO_4	Four	Not reported	Threshold for changes in the amplitude of alpha rhythms occurred when the sum of the fractional concentrations of the individual gases equaled 1.0 or more.	Kornienko, 1972

lowest NO_2 concentration causing impairment of dark adaptation was approximately 60 percent lower than the minimum concentration needed for odor perception. For SO_2, dark-adaptation impairment occurred at a concentration about 38 percent below that at which the odor was detected.

Kornienko (1972) determined odor-perception capabilities in a group of subjects exposed to individual gases or combinations of NO_2, SO_2, sulfuric acid aerosol, and ammonia. He reported also that the odor of any gas mixture was perceived whenever the sum of the fractional threshold concentrations of the component gases totaled one or more. Kornienko also investigated the effects of mixtures of these same gases on alpha rhythm in the brain. Again, he determined that the earliest decreases in amplitude, in response to combinations of the gases, occurred whenever the fractional threshold concentrations for the individual gases totaled one or more.

15.2.2 Pulmonary Function

15.2.2.1 Controlled Studies of the Effect of Nitrogen Dioxide on Pulmonary Function in Healthy Subjects--Controlled experimental studies in the laboratory situation have been mostly concerned with exposure to NO_2 alone although a few studies have considered effects during exposure to one or two additional air pollutants. These studies have generally been conducted on young healthy adults and/or on subjects suspected of being sensitive, e.g., individuals who have chronic respiratory problems. Controlled experimental studies on exposures of normal subjects to NO_2 are summarized in Table 15-3.

Nakamura (1964) determined the effect of exposure to combinations of NO_2 and sodium chloride aerosol (mean diameter 0.95 µm) on airway resistance (R_{aw}) measured by an interruption technique (Table 15-3). Two groups of seven and eight healthy subjects, 18 to 27 years old, were exposed for 5 minutes to 1,400 $\mu g/m^3$ sodium chloride aerosol alone. After resting for 10 to 15 minutes, individual subjects were exposed for 5 minutes to different NO_2 concentrations ranging from 5,600 $\mu g/m^3$ (3.0 ppm) to 7,500 (4 ppm). Nitrogen dioxide concentrations were measured by the Saltzman method. Each individual in each group showed increased R_{aw} after exposure to the NO_2 alone, but the sodium chloride aerosol alone exerted ho effect on airway resistance. Nitrogen dioxide alone at concentrations of 5,600 and 11,300 $\mu g/m^3$ (3.0 and 6.0 ppm) caused increases in R_{aw} of 16 and 34 percent, respectively, in the one subject tested at each concentration.

Von Nieding et al. (1970) reported, at the Second International Clean Air Congress, the results of exposures of 13 healthy subjects to an NO_2 level of 9,400 $\mu g/m^3$ (5.0 ppm) for 15 minutes (Table 15-3). Concentrations were measured by the colorimetric Saltzman method. At this level, a significant decrease in arterialized oxygen partial pressure (PaO_2) was induced, but the end expiratory oxygen partial pressure (PaO_2) remained unchanged. A reduced oxygen pressure in arterial blood would suggest a reduction in the transfer of oxygen from inspired air to blood in the lungs. The steady oxygen pressure in expired air would indicate that a constant supply of oxygen was delivered to the lungs. Together, the results suggest that NO_2 may have interfered with the transfer of oxygen from alveolar air to arterial blood. This

TABLE 15-3. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION IN CONTROLLED STUDIES OF HEALTHY HUMANS**

Concentration $\mu g/m^3$	Concentration ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
13,000	7.0	NO_2	Several	10-120 min.	Increased R_{aw}* in some subjects. Others tolerated 30,000 $\mu g/m^3$ (16 ppm) with no increase in R_{aw}.	Yokoyama, 1972
9,400	5.0	NO_2	11	2 hrs.	Increase in R_{aw}* and a decrease in $AaDO_2$* with intermittent light exercise. No enhancement of the effect when 200 $\mu g/m^3$ (0.1 ppm) O_3 and 13,000 $\mu g/m^3$ (5.0 ppm) SO_2 were combined with NO_2 but recovery time apparently extended.	Von Nieding et al., 1977
9,400	5.0	NO_2	16	15 min.	Significant decrease in DL_{CO}*	Von Nieding et al., 1973
9,400	5.0	NO_2	13	15 min.	Significant decrease in PaO_2* but end expiratory P_{O_2}* unchanged with significant increase in systolic pressure in the pulmonary artery.	Von Nieding et al., 1970
7,500 to 9,400	4.0 to 5.0	NO_2	5	10 min.	40% decrease in lung compliance 30 min. after exposure and increase in expiratory and inspiratory flow resistance that reached maximum 30 min. after exposure.	Abe, 1967
5,600	3.0	NO_2	1	5 min.	Increase in R_{aw}* compared to pre-exposure values (enhanced by NaCl aerosol).	Nakamura, 1964
11,300	6.0	NO_2	1	5 min.	More subjects were tested at higher exposures.	

(continued)

TABLE 15-3. (continued)

Concentration $\mu g/m^3$	Concentration ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
14,000	7.5	NO_2	16	2 hrs.	Increased sensitivity to a bronchoconstrictor (acetylcholine) at this concentration but not at lower concentrations.	
9,400	5.0	NO_2	8	14 hrs.	Increase in R_{aw} during first 30 min. that was reduced through second hour followed by greater increases measured at 6, 8 and 14 hrs. Also increased susceptibility to a bronchoconstrictor (acetylcholine).	Beil and Ulmer, 1976
4,700	2.5	NO_2	8	2 hrs.	Increased R_{aw} with no further impairment at higher concentrations. No change in arterial PO_2* pressure or PCO_2 pressure.	
1,880	1.0	NO_2	8	2 hrs.	No increase in R_{aw}.	Beil and Ulmer, 1976
1,880	1.0	NO_2	16	2 hrs.	No statistically significant changes in pulmonary function with the exception of small but statistically significant changes in FVC; see pp. 15-20.	Hackney, et al. 1978
1,300 to 3,800	0.7 to 2.0	NO_2	10	10 mins.	Increased inspiratory and expiratory flow resistance of approximately 50% and 10% of control values measured 10 mins. after exposure.	Suzuki and Ishikawa, 1965
1,150	0.6	NO_2	15	2 hrs.	No physiologically significant change in cardiovascular, metabolic, or pulmonary functions after 15, 30 or 60 mins. or exercise.	Folinsbee et al., 1978
1,880 to 3,760	1.0 to 2.0	NO_2	10	2½ hrs.	Alternating exercise and rest produced statistically significant decreases for hemoglobin hematocrit, and enythrocyte acetylcholinesterase.	Posin et al., 1978

(continued)

TABLE 15-3. (continued)

Concentration $\mu g/m^3$	Concentration ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
1,000	0.50	03	4	4 hrs.	With each group minimal alterations in pulmonary function caused by O_3 exposure. Effects were not increased by addition of NO_2 or NO_2 and CO to test atmospheres.	Hackney et al., 1975a,b,c
1,000 with	0.50	O_3				
560	0.29	NO_2				
1,000 with	0.50	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				
500	0.25	03	7	2 hrs.	Little or no change in pulmonary function found with O_3 alone. Addition of NO_2 or of NO_2 and CO did not noticeably increase the effect. Seven subjects included some believed to be unusually reactive to respiratory irritants.	Hackney et al., 1975a,b,c
500 with	0.25	O_3				
560	0.29	NO_2				
500 with	0.25	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				

(continued)

TABLE 15-3. (continued)

Concentration $\mu g/m^3$	Concentration ppm	Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
100 with	0.05	NO_2	11	2 hrs.	No effect on R_{aw}* or $AaDO_2$*; exposed subjects showed increased sensitivity of bronchial tree to a bronchoconstrictor (acetylcholine) over controls not exposed to pollutants.	Von Nieding et al., 1977
50 and	0.025	O_3				
300	0.11	SO_2				

* R_{aw} : airway resistance

$AaDO_2$: difference between alveolar and arterial blood partial pressure of oxygen

DL_{CO} : diffusion capacity of the lung for carbon monoxide

PaO_2 : arterial partial pressure of oxygen

PO_2 : partial pressure of oxygen

PCO_2 : partial pressure of carbon dioxide

** By descending order of lowest significant effect shown by each study.

increased difference between the alveolar and arterialized oxygen partial pressures (AaDO$_2$) was accompanied by a significant increase in systolic pressure in the pulmonary artery. However, the significance of the changes in A-aPO$_2$ are questionable in part due to the techniques employed and there is apparently some question as to the conditions present in their exposure chambers. Regardless of these questions, the potential of high NO$_2$ exposure to modify the delivery of oxygen to tissues remains unanswered. As noted later, no evidence for increased oxygen uptake during light to moderate exercise has been found.

Von Nieding et al. (1977) also exposed 11 healthy subjects, aged 23 to 38 years, to 9,400 $\mu g/m^3$ (5.0 ppm) NO$_2$ for 2 hours. Pulmonary function values, recorded prior to exposure, when the exposure was terminated, and 1 hour after exposure was terminated, were compared with similar values from control subjects exposed to clean air for 2 hours. Test subjects who underwent a regimen of intermittent light exercise during the testing period showed significant increases both in R_{aw} and in AaDO$_2$.

Von Nieding and co-workers (1973) observed a significant decrease in the lung's diffusion capacity for CO (DL_{CO}) in 16 healthy subjects resulting from a 15-minute inhalation of 9,400 $\mu g/m^3$ (5.0 ppm) NO$_2$ (Table 15-3). Abe (1967) found that concentrations of 7,500 to 9,400 $\mu g/m^3$ (4.0 to 5.0 ppm) for 10 minutes produced increases in both expiratory and inspiratory flow resistance in five healthy males; these increases reached a maximum 30 minutes after the end of exposure. Effective compliance (change in lung volume per unit change in air pressure), observed in this study 30 minutes after cessation of exposure, was decreased by 40 percent when compared with controls.

Increases in the inspiratory and expiratory flow resistance, observed in 10 healthy subjects exposed to NO$_2$ concentrations ranging from 1,300 to 3,800 $\mu g/m^3$ (0.7 to 2.0 ppm) for 10 minutes, were reported by Suzuki and Ishikawa (1965) (Table 15-3). Ten minutes after the exposure ceased, inspiratory resistance was increased 53 percent and expiratory resistance 13 percent. Information on variations in the results at different levels of exposure was not provided in this report. Since the exposure levels were reported by the authors to have varied during the studies, results are difficult to interpret. It is unlikely that concentrations of NO$_2$ would have varied extensively during a controlled exposure of 10 minutes duration. Results reported, however, were the averages of the responses of the 10 test subjects and may have reflected a response averaged across several exposure concentrations. The effect of NO$_2$ at a concentration of 9,400 $\mu g/m^3$ (5.0 ppm) was not increased by the addition of ozone (O$_3$) to the experimental atmosphere at a concentration of 200 $\mu g/m^3$ (0.1 ppm) or by adding 13,000 $\mu g/m^3$ (5.0 ppm) SO$_2$ to the NO$_2$/O$_3$ combination. When O$_3$ or the O$_3$ plus the SO$_2$ was added to the experimental atmosphere, the pulmonary function values, measured 1 hour after exposure was terminated, had not normalized as much as had the values in subjects exposed to NO$_2$ alone. Since subsequent measurements were not made, the only conclusion to be drawn from the study results is that recovery time following exposure to the multiple pollutants was delayed.

Yokoyama (1972) measured airway resistance in volunteers exposed to various concentrations of NO_2 for periods of 10 to 120 minutes. He measured increases in airway resistance at 13,200 $\mu g/m^3$ (7.0 ppm) and higher. He also recorded wide variations in individual sensitivity. Some volunteers tolerated concentrations as high as 30,000 $\mu g/m^3$ (16 ppm) with no increase in airway resistance. Because atropine effectively blocked the bronchoconstrictive effect of SO_2 but not of NO_2, this investigator suggested that the mechanism for the increase in airway resistance was unrelated to vagal stimulation.

Folinsbee et al. (1978) concluded from studies of three groups of five healthy males, ranging in age from 19 to 29 years, that no physiologically significant alterations in the measurements of pulmonary, cardiovascular, or metabolic factors were produced by 2-hour exposures to 1,150 $\mu g/m^3$ (0.61 ppm) NO_2 monitored by a continuous chemiluminescence technique (Table 15-3). Pulmonary measurements included: ventilatory volume (V_E); tidal volume (V_T); forced vital capacity (FVC); forced expiratory volume (FEV) at 1, 2, and 3 seconds, and forced expiratory flow (FEF) at 50 and 75 percent of vital capacity exhaled. Other measurements included oxygen (O_2) and carbon dioxide (CO_2) percentages in inspired and expired air, cardiac output, blood pressure, heart rate, steady state diffusion capacity of the lungs for carbon monoxide (DL_{CO}) and closing volume, with slow vital capacity (VC).

Hackney et al. (1978) found no statisically significant changes in pulmonary function in 16 healthy individuals exposed for 2 hours to 1,800 $\mu g/m^3$ (1.0 ppm) NO_2 with the exception of a marginal loss in forced vital capacity (FVC). The authors question the health significance of this latter small, but statistically significant change in FVC. That is, Hackney et al. (1978) stated:

> "As indicated, no changes with exposure were apparent except for a mean loss of 1.5% in FVC after the second exposure as compared to control. That this change represents other than a random variation is doubtful, due to its small size, its marginal statistical significance, and the relative large number of statistical comparisons being made."

Nitrogen dioxide was monitored by a continuous chemiluminescence analyzer and checked by the Saltzman method. Pulmonary functions measured included FVC, FEV, peak and maximum expiratory flow, closing volume (CV), R_{aw}, and others.

Beil and Ulmer (1976) exposed healthy volunteers (groups of 8 or 16) for 2 hours to 1,880, 4,700, 9,400, and 14,000 $\mu g/m^3$ (1.0, 2.5, 5.0, and 7.5 ppm) NO_2. Nitrogen dioxide concentrations were monitored by the continuous chemiluminescence method. An additional group was exposed for 2 hours to clean air. Following exposure to NO_2 at concentrations of 4,700 $\mu g/m^3$ (2.5 ppm) or above, these investigators measured significant increases in R_{aw} compared to the controls, but no decrease in PaO_2 or increase in $PaCO_2$. Airway resistance was not increased at a concentration of 1,880 $\mu g/m^3$ (1.0 ppm). Nitrogen dioxide concentrations of 9,400 or 14,000 $\mu g/m^3$ (5.0 or 7.5 ppm) did not produce significantly greater increases than did 4,700 $\mu g/m^3$ (2.5 ppm). In these healthy subjects, increased sensitivity to a bronchoconstrictor (0.5 percent acetylcholine for 1 minute inhalation at the rate of 0.12 liter per second) was observed after exposure for 2 hours to 14,000 $\mu g/m^3$ (7.5 ppm) NO_2 but not after exposure to 4,700 or 9,400 μg/m^3 (2.5 or 5.0 ppm). When the duration of exposure was increased from 2 to 14 hours, 9,400 μg/m^3 (5.0 ppm) NO_2 caused an initial increase in R_{aw} during the first 30 minutes. Airway resistance tended to return toward normal during the second hour. This was followed by even larger increases in R_{aw} measured after 6, 9, and 14 hours of exposure. The effect of exposure on two consecutive days was reversible, and R_{aw} measured 24 hours after initiation of exposure (10 hours after exposure was terminated) had returned to pre-exposure levels. Exposure of healthy subjects for 14 hours to 9,400 μg/m^3 (5.0 ppm) increased sensitivity to acetylcholine; the effects of longer exposures to lower concentrations were not tested.

Posin et al. (1978) exposed 10 subjects for 2.5 hours to filtered air on day one and on two consecutive days (days 2 and 3) to NO_2. The subjects alternated 15 minutes of exercise (double resting ventilation) and 15 minutes of rest. The ambient NO_2 levels were 1880 or 3760 μg/m^3 (1 or 2 ppm). Statistically significant decreases were observed for hemoglobin, hematocrit, and erythrocyte acetylcholinesterase. Glucose-6-phosphate dehydrogenase was elevated after the second exposure to 3760 μg/m^3 (2 ppm) NO_2 and levels of peroxidased red blood cell lipids were elevated after exposure to 3760 μg/m^3 (2 ppm). These investigators concluded that significant blood biochemical changes resulted from NO_2 inhalation. However, there are some questions as to the validity of these conclusions since there is considerable variability in the measurements that were made.

In contrast, Horvath and Folinsbee (1979) in a study on eight subjects exposed to 940 μg/m^3 (0.50 ppm) under a variety of ambient temperature conditions with one period of 30 minutes of exercise during the two hours failed to observe any changes in pulmonary functions. Also, Kerr et al. (1979) studied 10 normal healthy subjects exposed for 2 hours to 940 μg/m^3 (0.5 ppm) NO_2. One subject experienced the very mild symptom of slight rhinorrhea. Although the authors suggest that the changes reported in quasistatic compliance may be due to chance alone, there is uncertainty whether these changes were due to daily variation or to NO_2 exposure (see also page 15-27). No other significant alteration in pulmonary function resulted from the exposure.

When von Nieding and co-workers (1977) exposed 11 healthy subjects to a combination of NO_2 at 100 μg/m^3 (0.05 ppm), O_3 at 50 μg/m^3 (0.025 ppm), and SO_2 at 300 μg/m^3 (0.11 ppm) for 2 hours, no effect on R_{aw} or $AaDO_2$ was reported (Table 15-3). Exposure to this combination of pollutants did, however, produce what was interpreted by the investigators as dose-dependent increases over controls (not exposed to the pollutants) in the sensitivity of the bronchial tree to administered acetylcholine as measured by increases in R_{aw}. Constriction of the bronchi is a physiological alteration similar to that experienced by many individuals as asthma attacks. The suggestion provided by this study is that exposure to air pollutants may increase susceptibility to asthma attacks in some individuals.

Hackney et al. (1975a; 1975b; 1975c) exposed four healthy male volunteers to O_3 (1,000 μg/m^3; 0.5 ppm) and subsequently to mixtures of O_3 and NO_2 (560 μg/m^3; 0.3 ppm) or O_3, NO_2 and

CO (45,900 μg/m^3; 30 ppm) (Table 15-3). Volunteers were exposed for 4 hours plus about 1 additional hour during which several tests of pulmonary function were performed. The exposure regimen was designed to simulate exposure experienced during severe pollution episodes in Los Angeles on a summer day. The exposure time, however, was about twice that experienced in the ambient situation. Under these conditions, minimal alterations in pulmonary functions (FVC, FEV, CV, R_{aw} and others) were measured when test subjects were exposed to O_3 alone. These alterations were not increased by the additions of NO_2 or of NO_2 and CO. Another group of seven male volunteers, including some believed to be unusually reactive to respiratory irritants, was exposed under a similar protocol with an exposure time of 2 hours and to 500 μg/m^3 (0.25 ppm) O_3. Again, little or no change in pulmonary function was found with O_3 exposure alone, or with addition of NO_2 (560 μg/m^3; 0.3 ppm) or of NO_2 plus CO (45,900 μg/m^3; 30 ppm).

Schlipköter and Brockhaus (1963) determined, in three subjects, the effects of exposure to NO_2, carbon monoxide (CO), and SO_2 on pulmonary deposition of inhaled dusts. A suspension of homogenized soot (particle sizes 0.07 to 1.0 μm) was combined with either 9,000 μg/m^3 (4.8 ppm) NO_2, 55,000 μg/m^3 (50 ppm) CO, or 13,000 μg/m^3 (5.0 ppm) SO_2 and administered to experimental subjects by inhalation. The individual pollutant concentrations were the maximum acceptable concentrations in the Federal Republic of Germany. Pulmonary retention was determined by measuring the differences between the concentrations of dust in the inhaled and exhaled air. Under control conditions and with CO and SO_2 exposure, 50 percent of the dust was retained. Retention increased to approximately 76 percent when the dust was administered in an atmosphere containing 9,000 μg/m^3 (4.8 ppm) NO_2. Greater proportions of dust particles in the range of 0.3 to 0.8 μm were retained than were other size particles. This study is significant in that it demonstrates a potential additive or greater than additive mechanism that could operate in ambient situations involving significant NO_2 exposures of short duration. The study suggests that, as NO_2 concentrations in inhaled air increase, the response induced may result in respiratory retention of larger proportions of inhaled particles. If the particulate matter includes toxic materials, the additional impact on health could be significant.

Nakamura's (1964) studies on the interaction of NO_2 and sodium chloride aerosol indicated that sodium chloride aerosol had no influence on R_{aw}. When the sodium chloride aerosol (mean diameter 0.95 μm) was added to the exposure atmospheres, the increases in R_{aw} for the group were approximately 40 percent, about twice that produced by the gas alone. A sodium chloride aerosol comprised of smaller particles (mean diameter 0.22 μm) at 1,400 μg/m^3, in combination with the same concentrations of NO_2, produced no increase in R_{aw} over that caused by the gas alone. The consistent sequential methodology used in this study tends to reduce the credibility of this study; nevertheless, the fact that the final exposure challenge in the sequence increased the R_{aw} when the NaCl aerosol particles averaged 95 μm in diameter but did not increase R_{aw} when they averaged 22 μm in diameter indicates that when used in the same sequence the larger particles of NaCl enhanced the effect of NO_2 while the smaller particles did not.

Von Nieding and his co-workers (1970;1977) have conducted a number of studies of the effects of NO_2 on pulmonary function in healthy and bronchitic subjects. Some of the methods used for these studies differ from those employed routinely in the United States, and for this reason may not be directly comparable. For example, von Nieding measured R_{aw} during normal breathing using a body plethysmograph with a temperature compensation mechanism. Most American investigators have used constant-volume body plethysmographs and measure R_{aw} during panting (DuBois, 1956). Investigators in this country also measure arterial partial pressure of oxygen (PaO_2) in blood drawn directly from an artery, as opposed to a drop of blood obtained by pricking the ear lobe. In spite of the differences in technique, and the opinion offered here that the American methodology may be more accurate over the entire range of possible values, it is generally agreed that, in the hands of competent technicians, the methods used by von Nieding provide valid information on directional changes in airway resistance or changes in PaO_2.

Horvath and Folinsbee (1979) exposed eight young adults to either filtered air or 980 $\mu g/m^3$ (0.5 ppm) O_3 plus 940 $\mu g/m^3$ (0.5 ppm) NO_2 in filtered air under four different environmental conditions: (1) 25°C, 45% RH; (2) 30°C, 85% RH; (3) 35°C, 40% RH; and (4) 40°C, 50% RH. There were a total of eight exposures for each subject with a minimum of 1 week between exposures. During the exposures, the subject first rested for 60 minutes, then exercised for 30 minutes at 35-40% of his predicted maximum aerobic capacity and then rested for the final 30 minutes. Repeat tests were generally made on each subject at the same time of the day. The pulmonary responses to ozone alone were as found in previous studies by the same research group. No additive effect or interaction between ozone and nitrogen dioxide was observed.

In summary, studies on the effects of NO_2 on pulmonary functions in healthy volunteers (Table 15-3) indicate that exposure of 2 hours or less to concentrations of less than 4700 $\mu g/m^3$ (2.5 ppm) can induce increases in R_{aw} (Beil and Ulmer, 1976). The lowest concentration producing this effect is somewhat uncertain but is likely in the range of 1300 to 3800 $\mu g/m^3$ (0.7 to 2.0 ppm) (Suzuki and Ishikawa, 1965). Other changes in pulmonary function have been reported at higher concentrations. Exposure to a low-level mixture of NO_2, O_3, and SO_2 was also reported to increase sensitivity of healthy subjects to a bronchoconstrictor.

15.2.2.2 The Effects of Nitrogen Dioxide Exposure on Pulmonary Function in Sensitive Subjects--Subjects such as patients with asthma or chronic bronchitis have been studied by several investigators, as summarized in Table 15-4. For example, Von Nieding et al. (1973) exposed 14 patients with chronic bronchitis to NO_2 at a concentration of 9,400 $\mu g/m^3$ (5.0 ppm) for 15 minutes (Table 15-4). Alveolar partial pressures of oxygen measured before, during, and after inhalation of NO_2 were not altered significantly ($p > 0.05$). The earlobe arterialized blood partial pressure of O_2, however, decreased from an average of 102 x 10^2 to 95 x 10^2 pascals (76.6 to 71.4 torr) during exposure to the pollutant. Accompanying this was a significant increase in the difference of partial pressure of oxygen in alveoli (PAO_2) and

TABLE 15-4. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION IN CONTROLLED STUDIES OF SENSITIVE HUMANS

Concentration		No. of Subjects	Exposure Time	Effects	Reference
$\mu g/m^3$	ppm				
9,400	5.0	14 chronic bronchitics	60 mins.	No change in mean PAO_2*, during or after exposure compared with pre-exposure values, but PaO_2* decreased significantly in the first 15 mins. Continued exposure for 60 mins. produced no enhancement of effect.	Von Nieding et al, 1973
3,800 to 9,400	2.0 to 5.0	25 chronic bronchitics	10mins.	Significant decrease in PaO_2 and increase in $AaDO_2$* at 7,500 $\mu g/m^3$ (4.0 ppm) and above; no significant change at 3,800 $\mu g/m^3$ (2.0 ppm).	Von Nieding et al., 1971
940 to 9,400	0.5 to 5.0	63 chronic bronchitics	30 inhalations	Significant increase in R_{aw}* above 3,000 $\mu g/m^3$ (1.6 ppm); no significant effect below 2,800 $\mu g/m^3$ (1.5 ppm).	Von Nieding et al., 1971
940	0.5	10 healthy 7 chronic bronchitics 13 asthmatics	2 hrs.	1 healthy and 1 bronchitic subject reported slight nasal discharge. 7 asthmatics reported slight discomfort. Bronchitics and asthmatics showed no statistically significant changes for all pulmonary functions tested when analyzed as separate groups but showed small but statistically significant changes in quasistatic compliance when analyzed as a single group. See pp. 15-28.	Kerr, et al., 1978
190	0.1	20 asthmatics	1 hr.	Significant increase in SR_{aw}*. Effect of bronchoconstriction enhanced after exposure in 13 of 20 subjects. Neither effect observed in 7 of 20 subjects.	Orehek, et al., 1976

*PAO_2 : alveolar partial pressure of oxygen

R_{aw} : airway resistance

SR_{aw} : specific airway resistance

$AaDO_2$: difference between alveolar and arterial blood partial pressure of oxygen

PaO_2 : arterial partial pressure of oxygen in earlobe arterialized blood from an average of 34 x 10^2 to 43 x 10^2 pascals (25.5 to 32.3 torr). When exposure was continued for an additional 60 minutes, further significant disturbances of respiratory gas exchange were not observed.

Von Nieding et al. (1970;1971) also conducted a set of studies on NO_2 exposure with 88 chronic bronchitics (Table 15-4). Of these, 63 were tested for R_{aw} at 940 to 9400 μg/m^3 NO_2 (0.5 to 5.0 ppm) and 25 for PaO_2, PAO_2, $AaDO_2$, similar measurements for CO_2, and other parameters at 3700, 7500 and 9400 μg/m^3 (2.0, 4.0 and 5.0 ppm). Significant elevations in R_{aw} ($p < 0.1$) were seen after exposure to NO_2 concentrations of 3,000 μg/m^3 (1.6 ppm) and higher for 30 inhalations or approximately 3 minutes (Von Nieding et al., 1971). This increase became more pronounced at concentrations above 3,800 μg/m^3 (2.0 ppm), and disappeared completely below concentrations of 2,800 μg/m^3 (1.5 ppm). At levels of 7,500 to 9,400 μg/m^3 (4.0 to 5.0 ppm), subjects showed a significant decrease in PaO_2 and an increase in $AaDO_2$; no significant effect was found at 3800 μg/m^3 (2.0 ppm).

Kerr et al. (1978) studied the effects of 2 hours of exposure to NO_2 at a concentration of 940 μg/m^3 (0.5 ppm) on 7 chronic bronchitic and 13 asthmatic subjects. A 15-minute program of light to medium exercise on a bicycle ergometer was included in the exposure protocol. One of seven chronic bronchitics reported a slight nasal discharge associated with the exposure to NO_2. Seven of 13 asthmatics reported some evidence of slight discomfort, dyspnea, and headache with exercise. No significant changes were found in any of the pulmonary function parameters measured by spirometry, plethysmography, or esophageal balloon techniques when the groups were analyzed separately. When data for the two groups were analyzed together, small but statistically significant changes in quasistatic compliance were reported. However, the authors clearly stated:

> "The results of this investigation are in general negative, which is in itself useful. Although exposure to higher concentrations of NO_2 have been shown by others to alter function, and very high concentrations to result in significant damage to the respiratory system, it would appear that no significant alteration in pulmonary function is likely to result from a 2-hr exposure to 0.5 ppm NO_2 alone in normal subjects or subjects with chronic obstructive pulmonary disease. The few significant changes reported here may be due to chance alone."

Several studies have utilized a challenge with acetylcholine in order to further clarify the pulmonary responses to nitrogen dioxide. Von Neiding et al. (1977) suggested that there was an increased sensitivity of the bronchial tree to administered acetycholine in subjects exposed to a very low level of NO_2. Beil and Ulmer (1976) reported an increased sensitivity to acetylcholine after a 2-hour exposure to 14,100 μg/m^3 (7.5 ppm) NO_2 but not after 2-hr exposures to 4700 or 9400 μg/m^3 (2.5 or 5.0 ppm) NO_2. They further found an increased sensitivity to acetylcholine in subjects exposed for 14 hours to 9400 μg/m^3 (5.0 ppm) NO_2.

Orehek et al. (1976) studied the effects of low levels of NO_2 exposure on the bronchial sensitivity of asthmatic patients to carbachol, a bronchoconstricting agent. In this study the carbachol was used to induce a response in asthmatics similar to the response occurring when they are exposed to particular natural agents to which they are sensitive. Nitrogen dioxide concentrations were monitored by the Saltzman method. For 20 asthmatics, dose-response curves were developed for changes in specific airway resistance (SR_{aw}) as a result of the subjects inhaling carbachol after a 1-hour exposure to clean air and, on other occassions, after a 1-hour exposure to 190 $\mu g/m^3$ NO_2 (0.1 ppm). Following NO_2 exposure alone, slight or marked increases in SR_{aw} were observed in only 3 of 20 asthmatic test subjects; however, NO_2 exposure at 0.1 ppm enhanced the effect of the bronchoconstrictor in 13 of 20 subjects. The mean dose of carbachol producing a two-fold (100%) increase in SR_{aw} in the 13 sensitive subjects was significantly decreased from 0.66 mg to 0.36 mg as a result of NO_2 exposure. Seven of the asthmatic subjects showed neither an increase in R_{aw} in response to the exposure to NO_2 alone nor an enhanced effect of NO_2 on carbachol-induced bronchoconstriction.

Unfortunately, the reported statistical significance of some of the data is rendered difficult to interpret, likely due to the small sample size. For example, the mean dose of carbachol producing a 100 percent increase in R_{aw} was reported to be 0.36 mg for 7 non-responders and 0.66 mg for 13 responders, a non-statistically significant difference. On the other hand, the mean doses of carbachol producing a 100 percent increase in R_{aw} for 13 responders before and after the NO_2 exposure were 0.66 mg and 0.36 mg, respectively; but, in this instance, the same absolute difference in means was reported to be statistically significant. Similarly, a 15 percent difference in mean R_{aw} for 13 responders before and after NO_2 exposure alone was reported to be statistically significant, but a 20 percent difference in the initial mean R_{aw} between responders and non-responders was reported to not be significant.

The results of this study are of interest because they are suggestive of possible bronchoconstrictive responses being produced in some asthmatics by very low concentrations of NO_2. These results, however, do not provide conclusive evidence of adverse responses attributable to NO_2 exposure, especially in view of some of the reported statistically significant NO_2-induced changes not being markedly different in average magnitude to changes in R_{aw} apparently due to individual variations in lung function in the absence of NO_2. The testing protocol used in the study was an extremely sensitive one, i.e., employing known sensitive subjects, a potent pharmacologic agent, and measurements of flow resistance. It is obvious from the reported responses of the 20 subjects to the test regimen that only three showed measurable variations as a result of exposure to NO_2 alone. However, the mean of measurements of R_{aw} in 13 responders to the carbachol treatment was significantly higher after the NO_2 exposure than it had been prior to exposure. The criticism of this reported change was that the comparisons of R_{aw} were made in subjects selected not at the time of NO_2 exposure, but after the fact, following the carbachol exposure. There was no report of the initiation of an asthma attack, or even wheezing, or lack of such symptoms in any subject, as a result of the combined insults of carbachol and NO_2, clouding interpretation of how the observed effects might relate to asthma attacks under ambient conditions. The study may have health implications, however, since it suggests that those asthmatics whose illness results from vagal stimulation might be predisposed to have more severe attacks (once induced by other agents) as a result of also breathing NO_2. However, such a suggestion (that the effects of a potent vagal stimulus may be increased by the inhalation of low levels of NO_2 seems to be at odds with the report of Yokoyama (1972) discussed earlier, who found that atropine blocked the bronchoconstrictor effect of SO_2 but not NO_2 and, for this reason, concluded that NO_2 did not act by stimulating the vagus nerve. It thus remains to be determined as to what concentrations of NO_2 may produce significant broncoconstriction or other pulmonary mechanical effects in asthmatics under ambient exposure conditions.

Another suggestion that measurably greater impact of exposure to NO_2 occurs in highly susceptible individuals is obtained from the studies of Barter and Campbell (1976). These investigators studied a group of 34 subjects with mild bronchitis and showed that decreases in $FEV_{1.0}$, over a period of 5 years, were related to the subjects' degree of reactivity to the bronchoconstrictor, methacholine. Even minimal cigarette smoking, a source of significant concentrations of nitrogen oxides (Norman and Keith, 1965), led to ventilatory deterioration, which the investigators believed to be serious when methacholine reactivity was high. However, heavy smoking had little effect on ventilatory function when reactivity to methacholine was slight. It is not certain that the effective material in the cigarette smoke causing the impairments in ventilatory function was NO_2, although this seems to be a good possibility. It also is not known how these study results relate to ambient NO_2 exposures of individuals who are highly reactive to methacholine.

Thomas et al. (1972) showed no effect of exposure to NO_2 at concentrations of 940 to 6,580 $\mu g/m^3$ (0.5 to 3.5 ppm) on histamine concentrations in sputum or on total sputum weight, in five healthy subjects, or four patients reported to have chronic respiratory disease.

In summary, studies of NO_2 effects on pulmonary function in potentially susceptible population groups show that, in persons with chronic bronchitis, concentrations of 9,400 $\mu g/m^3$ (5.0 ppm) produce decreases in PaO_2 and increases in $AaDO_2$, whereas exposures to concentrations of NO_2 above 2,800 $\mu g/m^3$ (1.5 ppm), for periods considerably less than 1 hour, produce significant increases in R_{aw} ($p < 0.1$). Thus, results from bronchitic individuals and healthy individuals differ very little. In contrast, in one study, exposures to 190 $\mu g/m^3$ (0.1 ppm) NO_2 for 1 hour were reported to have increased mean R_{aw} in 3 of 20 asthmatics and to have enhanced the effects of a bronchoconstrictor in 13 of the same 20 individuals. However, in another study, no measurements of pulmonary function were significantly altered in 13 asthmatics as a result of 2 hours of exposure to 940 $\mu g/m^3$ (0.5 ppm) NO_2. Thus, whereas NO_2 exposures sufficient to produce increased R_{aw} in healthy individuals or those with symptoms of chronic respiratory illness may indeed produce much greater and more severe responses in other highly susceptible segments of the population (e.g., asthmatics), controlled human exposure studies to date do not convincingly demonstrate pulmonary function changes in susceptible population groups at NO_2 exposure levels below those affecting normal, healthy adults.

15.3 EPIDEMIOLOGICAL STUDIES

Epidemiological studies of the effects of community air pollution are complicated because there are complex varieties of pollutants present in the ambient air that exposed populations breathe. Thus, the most that can usually be demonstrated by such studies is that an association exists between observed health effects and a mixture of air pollutants. In order for inferences to be drawn regarding likely causal relationships between any individual pollutant present in such mixtures and observed effects, consistent associations must be demonstrated between variations in exposures to the pollutant and particular types of effects under a variety of circumstances (of course with potentially covarying or confounding variables having been adequately controlled for or taken into account in the analysis of study results).

In attempting to demonstrate qualitative or quantitative relationships between NO_2 and health effects, presently available epidemiological studies have been notably hampered by difficulties in defining actual exposure levels or durations for study populations. For example, many NO_2-related epidemiological studies conducted prior to 1970 are of questionable validity due to a number of instrumental and analytical problems inherent in the air monitoring technique (Jacobs-Hochheiser method) typically employed for measuring atmospheric concentrations of NO_2. Still other study results can be questioned on the basis of how representative reported aerometric results are of actual NO_2 exposures of study populations. For these reasons, the contributions of pre-1970 community air pollution studies to knowledge concerning NO_2 exposure effects are highly limited at this time and are, therefore, not discussed in detail here.

Rather, only selected pre-1970 studies and other relevant post-1970 studies are discussed below, together with notation (as appropriate) of certain major problems affecting interpretation or acceptance of their results. The outdoor pollution studies thusly assessed can be conveniently divided into the following categories: (1) those evaluating potential associations between NO_2 exposures and diminished pulmonary functions; and (2) those evaluating possible increased risks for acute or chronic respiratory diseases in response to NO_2 exposures.

In addition to the outdoor ambient air pollution studies, other epidemiological evaluations of the effects of indoor air pollution are also discussed, with particular emphasis on possible relationships between indoor exposures to NO_2 and respiratory diseases in children.

15.3.1 Effects of NO_2 on Pulmonary Function

Among the better known studies evaluating NO_2-related health effects are those conducted by Shy et al. (1970a,b;1973) on schoolchildren during 1968-69 in Chattanooga, Tennessee. Shy et al. (1970a) reported that values for 0.75-second forced expiratory volume were lower in those children living in areas of apparently high NO_2 concentrations than for children living in areas with lower NO_2 concentrations. However, measurements of NO_2 concentrations used in this study were done by means of the Jacobs-Hochheiser method*, which was subsequently found to be unreliable and not acceptable for deriving quantitative estimates of NO_2 levels present in the 1968-69 Chattanooga study areas. In view of some overlap between NO_2 levels reported for certain monitoring sites in the "high" NO_2 pollution study areas and NO_2 levels for some monitoring sites in the lower NO_2 pollution study areas (based on the original Jacobs-Hochheiser readings), even qualitative comparisons of effects observed between the study areas are of questionable value. This is especially true given that differential effects observed with pulmonary function tests were stated to be small (although statistically significant) and showed inconsistencies during the testing period.

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*The Jacobs-Hochheiser technique has been withdrawn by EPA and replaced by a new Federal Reference Method (chemiluminescence) and other equivalent methods (see Chapter 7).

Several other community health epidemiological studies in various geographic areas have attempted to provide quantitative assessments of pulmonary function changes in relationship to ambient air NO_2 levels. These studies, using a more acceptable NO_2 measurement technique (the Saltzman method), are summarized in Table 15-5. For example, Speizer and Ferris (1973b) administered pulmonary function tests to 128 traffic policemen in urban Boston and to 140 patrol officers in nearby suburban areas (Table 15-5) but found no differences in pulmonary function between the two study groups. Mean 24-hour NO_2 concentrations determined from 1-hour sampling data, measured by the Saltzman technique, were 100 $\mu g/m^3$ (0.055 ppm) in the downtown urban area, and 75 $\mu g/m^3$ (0.04 ppm) in the suburban area (Speizer and Ferris, 1973a). Sulfur dioxide levels averaged 92 $\mu g/m^3$ (0.035 ppm) in the city and 36 $\mu g/m^3$ (0.014 ppm) in the suburban area.

Cohen et al. (1972) also found no differences in the results of several ventilatory tests, including spirometry and flow-volume curves, for nonsmoking adults living in the San Gabriel Valley of the Los Angeles basin and similar nonsmokers from San Diego (Table 15-5). The average NO_2 concentration in the Los Angeles basin was 96 $\mu g/m^3$ (0.05 ppm) based on the Saltzman method. The ninetieth percentile of the daily averages in this area, i.e., the level exceeded only 10 percent of the time, was 188 $\mu g/m^3$ (0.1 ppm). In San Diego, the average and ninetieth percentile were, respectively, 43 and 113 $\mu g/m^3$ (0.02 and 0.06 ppm) based on Saltzman method measurements.

Linn et al. (1976) performed a variety of pulmonary function tests during the summer and winter seasons on 205 office workers of both sexes in Los Angeles and 439 similar individuals in San Francisco. Additional information about respiratory symptoms was obtained by means of personal interviews. This study was undertaken primarily to determine the effects of oxidant air pollution, but information regarding NO_2 was provided as well. Most results of FEV, single breath N_2 tests, and interviews showed no differences between cities. Los Angeles women did report nonpersistent cough and phlegm more often than did San Francisco women, and smokers in both cities showed greater changes in pulmonary function than did nonsmokers. Median hourly oxidant values (primarily O_3) were 0.07 and 0.02 ppm in Los Angeles and San Francisco, respectively. Ninetieth percentile oxidant values were 0.15 and 0.03 ppm. The median hourly NO_2 concentrations based on the Saltzman method were 130 and 65 $\mu g/m^3$ (0.07 and 0.035 ppm), respectively, for Los Angeles and San Francisco. The 90th percentile hourly NO_2 concentrations were 250 and 110 $\mu g/m^3$ (0.13 and 0.06 ppm) for Los Angeles and San Francisco, respectively.

TABLE 15-5. QUANTITATIVE COMMUNITY HEALTH EPIDEMIOLOGICAL STUDIES ON EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION

Measure	NO_2 Exposure Concentrations µg/m^3	NO_2 Exposure Concentrations ppm	Study Population	Effect	Reference
High exposure group:					
Annual mean 24-hr concentrations	96	0.051	Nonsmokers Los Angeles (adult)	No differences in several ventilatory measurements including spirometry and flow volume curves	Cohen et al., 1972
90th percentile	188	0.01			
Estimated 1-hr maximum[a]	480 to 960	0.26 to 0.51			
Low exposure group:					
Annual mean 24-hr concentrations	43	0.01	Nonsmokers San Diego (adult)		
90th percentile	113	0.06			
Estimated 1-hr maximum[a]	205 to 430	0.12 to 0.23			
Mean "annual"[b] 24-hr concentrations: high exposure area	103 + 92 SO_2	0.055 + 0.035 SO_2	Pulmonary function tests administered to 128 traffic policemen in urban Boston and to 140 patrol officers in nearby suburban areas.	No difference in various pulmonary function tests.	Speizer and Ferris, 1973a,b Burgess et al., 1973
low exposure area	75 + 36 SO_2	0.04 + 0.014 SO_2			
1-hr mean: high exposure area	260 to 560	0.14 to 0.30			
low exposure area	110 to 170	0.06 to 0.09			

TABLE 15-5. (continued)

Measure	NO_2 Exposure Concentrations $\mu g/m^3$	ppm	Study Population	Effect	Reference
Los Angeles:					
Median hourly NO_2	130	0.07	205 office workers in Los Angeles	No differences in most tests. Smokers in both cities showed greater changes in pulmonary function than non-smokers.	Linn, et al., 1976
90th percentile NO_2	250	0.13			
Median hourly O_x		0.15			
90th percentile O_x		0.15			
San Francisco:					
Median hourly NO_2	65	0.35	439 office workers in San Francisco		
90th percentile NO_2	110	0.06			
Median hourly O_x		0.02			
90th percentile O_x		0.03			
1-hr concentration at time of testing (1:00 p.m.)	40 to 360	0.02 to 0.19	20 school children 11 years of age	During warmer part of the year (April-October) NO_2, SO_2 and TSP* significantly correlated with $\dot{V}_{max}$* at 25% and 50% FVC* and with specific airway conductance. Temperature was the factor most clearly correlated with weekly variations in specific airway conductance with $\dot{V}_{max}$ at 25% and 50% FVC. Significant correlation between each of four pollutants (NO_2, NO, SO_2 and TSP) and $\dot{V}_{max}$ at 25% and 50% FVC; but no clear delineation of specific pollutant concentrations at which effects occur.	Kagawa and Toyama, 1975

[a] Estimated at 5 to 10 times annual mean 24-hour averages

[b] Mean "annual" concentrations derived from 1-hour measurements using Saltzman technique

*$FEV_{0.75}$: Forced expiratory volume, 0.75 seconds

$\dot{V}_{max}$: Maximum expiratory flow rate

FVC : Forced vital capacity

TSP : Total suspended particulates

In a Japanese investigation, relationships of ambient temperature and air pollutants (NO_2, NO, O_3, hydrocarbons, SO_2, and particulate matter) to weekly variations in pulmonary function in 20 school children, 11 years of age, were studied in Tokyo by Kagawa and Toyama (1975) and Kagawa et al. (1976). Of all the factors assessed, temperature was most closely correlated ($p < 0.05$) with variations in specific airway conductance, and with maximum expiratory flow rate ($\dot{V}_{max}$) at 25 and 50 percent of FVC. In children believed by the investigators to be sensitive to air pollution, a significant negative correlation was observed between exposure to O_3 and specific airway conductance; other negative correlations were found between NO_2 (as measured by the Saltzman technique), NO, SO_2, and particulate matter, with $\dot{V}_{max}$ at 25 percent or 50 percent FVC. At high outdoor temperatures NO_2, SO_2, and particulate matter were significantly correlated (negatively) with both $\dot{V}_{max}$ at 25 percent or 50 percent FVC, and specific airway conductance ($p < 0.05$).

In the ambient situation, however, the above effects were not associated with NO_2 alone, but with the combinations of air pollutants, including SO_2, particulate matter, and O_3. During the period of high outdoor temperatures, correlations between lung function and NO_2 concentrations were calculated using the pollutant level in the ambient air at the time of testing (1:00 p.m.). These hourly NO_2 values ranged from 40 to 360 $\mu g/m^3$ (0.02 to 0.19 ppm), but the data reported afforded no quantitative estimation of specific NO_2 levels that might have been associated with the occurrence of pulmonary function decrements. The authors (Kagawa et al., 1976) noted that: "From the relationship between NO_2 and V_{max} at 50% FVC in subject No. 16, who showed the highest correlation, it seems that NO_2 started having a significant effect above a concentration of 4 pphm." The basis for this statement, however, is not obvious from the published (Kagawa and Toyama, 1975; Kagawa et al., 1976) data or statistical analyses, which do not appear to provide sufficient bases for estimating NO_2 air concentrations associated with pulmonary function decrements.

In other Japanese studies measurements of pulmonary function in employees exposed to diesel exhausts in railroad tunnels were reported by Mogi et al. (1968) and by Yamazaki et al. (1969). Results of pulmonary function tests were compared with similar results from employees in other situations in which exposure was classified as medium, light, or "no-pollution." Mean NO_2 concentrations, measured by the Saltzman method, ranged from 300 to 1,130 $\mu g/m^3$ (0.16 to 0.60 ppm). Highest measured NO_2 concentrations ranged from 340 to 3,000 $\mu g/m^3$ (0.18 to 1.60 ppm). Test results [VC, $FEV_{1.0}$, maximal flow rate (MFR) and mid-maximal flow rate (MMFR)] from 475 employees were highest in those working in "no-pollution" areas. Results obtained on the remaining subjects showed a decrease in pulmonary function which did not correlate with the NO_2 concentrations in their work areas.

Results of epidemiological studies on the effects of outdoor ambient air NO_2 exposures provide no consistent indication that the mean concentrations of NO_2 or of NO_2 in combination with other pollutants listed in Table 15-5 had any significant effects on lung function in the exposed populations. One study did show some apparent associations between V_{max} or specific airway conductance and concentrations of NO_2 and other pollutants at the time of testing, but the contributions of individual air pollutants were difficult to disentangle and appeared to be less than those attributable to temperature variations.

15.3.2 Effects of NO_2 on Acute Respiratory Illness

15.3.2.1 Effects Associated with Ambient Exposures--Only a few community epidemiological studies of outdoor NO_x exposures have been reported as demonstrating associations between ambient air levels of NO_x compounds and measurable health effects. However, methodological problems with the studies preclude acceptance of the reported results as clear evidence for increases in acute respiratory illness being due to NO_x exposures.

Shy and co-workers (Shy, 1970; Shy et al., 1970b; 1973) studied the effects of community exposure to NO_2 in residential areas of Chattanooga on respiratory illness rates in families. The distances of three study communities from a large point source of NO_2 resulted in an apparent gradient of exposure over which the illness rates were determined. The incidence of acute respiratory disease was assessed at 2-week intervals during the 1968-69 school year and the respiratory illness rates adjusted for group differences in family size and composition were reported to be significantly higher for each family segment (mothers, fathers, children) in the high-NO_2 exposure neighborhood than in the intermediate- and low-NO_2 areas. However, NO_2 concentrations were determined by the Jacobs-Hochheiser method, and this method has since been shown to be unreliable (Hauser and Shy, 1972; See Chapter 7 for more details). Meaningful quantitative estimates of population NO_2 exposures were therefore not available for the study areas; also, overlaps in reported NO_2 levels between "high" NO_2 area monitoring sites and those for lower NO_2 study areas make qualitative comparisons between the study areas somewhat problematic. In addition, no basis was provided by which the relative contribution of NO_2 exposures could be separated from those of other pollutants present in the study areas. Additional acute respiratory disease rate data for the same Chattanooga study areas were collected in 1972-1973, by which time NO_2 levels differed little between the study areas as measured by the Saltzman technique. Preliminary analyses of these data were reported by Shy and Love (1979), but the analyses reported in their 1979 paper for Jan.-May, 1973 were later found to actually apply to data collected in the fall of 1972 and the analyses reported for both 1972 and 1973 included data misclassified by subject age (personal communication from Shy).

In an earlier retrospective study in the same Chattanooga areas, Pearlman et al. (1971) investigated the frequency of lower respiratory disease among first- and second-grade school children and among children born between 1966 and 1968. Responses on 14 percent of the health status questionnaires were validated against physician and hospital records, with overall accuracy of parental reporting of illness episodes being 70.5, 67.9, and 90.0 percent for bronchitis, croup, and pneumonia, respectively. No significant study area differences in illness rates were found for croup, pneumonia, or a combined "any lower respiratory disease" category in pre-school infants or schoolchildren; nor did illness rates for these illness categories follow the reported pollutant gradient for any age group classified by period (1,2, or 3 yrs) of residence in the respective study areas. Similarly, illness rates for bronchitis failed to follow the reported NO_2 exposure gradient (except for schoolchildren of 3 yr residence), with intermediate area bronchitis rates otherwise generally exceeding those determined for the children in the high NO_2 area. Only bronchitis rates in schoolchildren who had lived in the area for 3 or more years followed the exposure gradient and were found to be significantly higher than for children from the low NO_2 area. The validity of this reported difference, however, appears to be questionable in view of: (1) its internal inconsistency with the other study results reported; (2) the only moderately high accuracy (70.5 percent) of reporting of bronchitis rates in comparison to the accuracy (90 percent) for pneumonia rates; and (3) the reported inability of parents to date precisely the occurrence of bronchitis episodes, making it impossible to separate such illnesses occurring before residence in the exposure areas from those occurring thereafter. Also, the same difficulties in defining the reported NO_2 exposure gradient (presumably based on use of the Jacobs-Hochheiser NO_2 method) for the study areas used in this study likely apply as noted above for the Shy et al. 1968-69 Chattanooga studies.

Polyak (1968) reported 44 percent more health clinic visits for respiratory, visual, nervous symptoms, and skin disorders by residents living within 1 kilometer of a Soviet Union chemical works than by residents living more than 3 kilometers from the factory complex. The study subjects, none of whom worked at the chemical plant, were exposed to a combination of pollutants. Concentrations reported were: NO_2, 580 to 1,200 $\mu g/m^3$ (0.31 to 0.64 ppm); SO_2, 225 $\mu g/m^3$ (0.09 ppm); and sulfuric acid, 400 $\mu g/m^3$ (0.1 ppm). The report also indicated that a NO_2 concentration of 1,600 $\mu g/m^3$ (0.85 ppm), combined with high concentrations of SO_2 and sulfuric acid, occurred 1 kilometer from the plant. However, no information was provided on the methods used to measure NO_2 or by which to assess the relative contribution of NO_2 exposures to the reported health effects.

In another study from the Russian literature, Giguz (1968) studied illness rates and other factors in 16- to 19-year-old vocational trainees in the Soviet Union. Individuals training in fertilizer or chemical manufacturing plants (N=145) were compared with 85 individuals of the same age not exposed to pollutants found in the manufacturing plants. Exposure concentrations of NO_2 and ammonia, gases expected by the investigators to occur in highest concentrations in this situation, did not exceed the maximum permissible concentrations (average daily mean: for NO_2, 100 $\mu g/m^3$ or 0.053 ppm in 1964) in the U.S.S.R. (Nikolaeva, 1964). Subjects were exposed to the pollutants for 3 hours a day for 150 days during the first year of training, and for 6 hours a day for 200 days during the second year. During the second year of training, exposed individuals were reported to have an increased incidence of acute respiratory disease and increased serum levels of beta-lipoproteins, cholesterol, and albumin. However, this report lacks important information related to air pollutant sampling frequencies and methods, making it difficult to evaluate the reliability of quantitative estimates of NO_2 levels associated with reported health effects.

Petr and Schmidt (1967) studied acute respiratory illness among Czechoslovakian schoolchildren living near a large chemical complex. They found a disease incidence twice that observed in children of the same age living in a low-exposure community of similar socioeconomic characteristics. A greater number of hypertrophied tonsils and cervical lymph nodes also were found in children from two towns, each having high pollution but differing with respect to relative concentrations of NO_2 and SO_2. Children from the area having the lower concentrations of NO_x had much lower lymphocytogram values and higher indices of proliferation and differentiation of monocytes. The clinical significance of these differences is not known and the results of the Petr and Schmidt (1967) study are difficult to interpret because data on concentrations of NO and NO_2 individually were not presented. Nor was information provided on the sampling frequency or metholog for NO or NO_2 measurements or on levels of other pollutants such as acid aerosols, particulate nitrates, sulfates, or total suspended particulate matter. It is probable that such a variety of pollutants was present around a large chemical complex, but no basis was provided by which to assess the relative contribution to reported health effects of NO_x compounds from among other air pollutants present.

15.3.2.2 Effects Associated With Indoor Exposures

15.3.2.2.1 Tobacco smoking studies. Tobacco smoke is a major source of nitrogen oxides, and may include significant concentrations of NO_2. Norman and Keith (1965) demonstrated that the concentration of NO may vary between 492×10^3 and 1.23×10^6 $\mu g/m^3$ (400 and 1,000 ppm) in cigarette smoke depending on the type of filter included and the extent to which the cigarette is smoked, i.e., the last puff contained higher concentrations than did the fourth puff. Often NO_2 could not be measured in the cigarette smoke but, in other instances, concentrations as high as 47,000 $\mu g/m^3$ (25 ppm) were found. In confined spaces such as meeting rooms, concentrations of NO_2 might build up to potentially hazardous levels, when several individuals are smoking. Smoking then may represent a significant source of exposure for nonsmokers as well as smokers.

Many adverse health effects have been associated with smoking, particularly cigarette smoking. It has not been possible, however, to implicate conclusively any single factor in cigarette smoke as the one primarily responsible for the effects observed. Other major pollutants in cigarette smoke include CO and tars. Each of these materials, in experimental situations, causes a fairly unique type of effect. Consequently, even though no specific adverse effects can be attributed conclusively only to the NO_2 in cigarette smoke, the increases in some adverse health parameters point strongly in this direction. Specific health effects possibly related to exposure to NO_2 in cigarette smoke include increased acute respiratory illnesses or prevalence of chronic respiratory disease.

15.2.2.2.2 Epidemiological Studies of Gas Combustion Products. Combustion of natural or manufactured gas within confined spaces leads to increases in NO_2 concentrations. Although some CO and other pollutants may also be produced by gas-burning appliances, the relative efficiencies of the combustion process cause the largest quantities of NO_2 to be produced when CO production is lowest. This tends to suggest that increased health effects, if observed in homes with gas stoves, are more likely the result of NO_2 exposure than CO exposure; however, effects due to exposure to other potentially toxic products of gas combustion cannot be ruled out.

Social characteristics of family life, as well as the size and type of housing, also affect exposure parameters for those people living in homes with pollution produced by gas appliances or other sources. For example, a home with a small kitchen containing a gas stove may represent a greater hazard for individuals who spend much time in the kitchen, especially if the social custom is to heat as little of the home as possible, and most or all of the family spend considerable time in or near the kitchen. In such homes, relatively frequent (almost continuous) exposure periods to gas stove pollutants may be experienced by family members. In other homes, however, such as those with central heating, family members may spend much less time in the kitchen and, therefore, experience less exposure to gas combustion products.

Several studies show that homes with gas stoves typically have higher NO_2 levels than those with electric stoves. For example, Wade et al. (1975) reported that, over a 2-week period, the average concentration of NO_2 in kitchens with gas stoves exceeded 94 $\mu g/m^3$ (0.05 ppm) and that in different parts of the house, the concentrations fluctuated with use of the stoves. Nitrogen dioxide was monitored by a chemiluminescence method. Average levels of 280 $\mu g/m^3$ (0.15 ppm) NO_2 for 2 hours were measured in the kitchen. Other studies of NO_2 concentrations in homes during the preparation of meals have demonstrated that gas stoves produce NO_2 concentrations usually within the range of 470 to 1100 $\mu g/m^3$ (0.25 to 0.6 ppm) in the immediate vicinity of the stove (Mitchell et al., 1974; U.S. Environmental Protection Agency, 1976). The highest level recorded was 1,880 $\mu g/m^3$ (1.0 ppm). The high concentrations are significantly reduced within 2 hours after the stove is turned off. The specific time for reduction of peak concentrations by any given percentage after the cessation of stove usage, however, depends upon such parameters as the presence or absence of ventilation devices and the details of interior architecture. Other studies also indicate NO_2 is higher in homes using gas than those using electricity (Melia et al., 1978; Goldstein et al., 1979; Spengler et al., 1979).

Observations of elevated NO_2 and other air pollutant levels in gas stove homes in comparison to electric stove homes have prompted epidemiological investigations of possible health effects associated with the higher levels of indoor air pollutants in homes using gas stoves. The results of such epidemiological studies are summarized in Table 15-6.

TABLE 15-6. EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE IN THE HOME ON LUNG FUNCTION AND THE INCIDENCE OF ACUTE RESPIRATORY DISEASE IN EPIDEMIOLOGY STUDIES OF HOMES WITH GAS STOVES

Pollutant[a] Studies of Children	NO_2 Concentration µg/m³	NO_2 Concentration ppm	Study Population	Effects	Reference
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study		2554 children from homes using gas to cook compared to 3204 children from homes using electricity. Ages 6-11	Proportion of children with one or more respiratory symptoms or disease (bronchitis, day or night cough, morning cough, cold going to chest, wheeze, asthma) increased in homes with gas stoves vs. electric stove homes (for girls $p \simeq 0.10$; boys not sig.) after controlling for confounding factors.	Melia et al., 1977
NO_2 plus other gas stove combustion products	NO_2 concentration not measured in same homes studied		4827 children ages 5-10	Higher incidence of respiratory symptoms and disease associated with gas stoves (for boys $p \sim 0.02$; girls $p \simeq 0.15$) after controlling for confounding factors	Melia et al., 1979
NO_2 plus other gas stove combustion products	Kitchens: 9-596 (gas) 11-353 (elec) Bedrooms: 7.5-318 (gas) 6 - 70 (elec) (by triethanolamine diffusion samplers)	 0.005-0.317 0.006-0.188 0.004-0.169 0.003-0.037	808 6- and 7-year-old children	Higher incidence of respiratory illness in gas-stove homes ($p \simeq 0.10$). Prevalence not related to kitchen NO_2 levels, but increased with NO_2 levels in bed rooms of children in gas-stove homes. Lung function not related to NO_2 levels in kitchen or bedroom	Florey et al., 1979 Companion paper to Melia et al., 1979; Goldstein et al., 1979
NO_2 plus other gas stove combustion products	95 percentile of 24 hr avg in activity room 39 - 116 µg/m³ (.02 - .06 ppm) (gas) vs. 17.6 - 95.2 µg/m³ (.01 - .05 ppm) (electric). Frequent peaks ≃ 1100 µg/m³ (0.6 ppm), max peak ~ 1880 µg/m³ (1.0 ppm) 24 - hr by modified sodium arsenite; peaks by chemiluminescence		8,120 children 6-10 yrs old in 6 different communities with data collected on lung function and history of illness before the age of 2	Significant association between history of serious respiratory illness before age 2 and use of gas stoves ($p < .01$) and, also, between lower FEV, FVC levels and use of gas stoves ($p < .01$)	Speizer et al., 1980 Spengler et al., 1979

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TABLE 15-6 (continued)

Pollutant[a]	NO_2 Concentration $\mu g/m^3$ ppm	Study Population	Effects	Reference
NO_2 plus other gas stove combustion products	Sample of households 24 hr avg: gas (.005 - .11 ppm); electric (0 - .06 ppm); outdoors (.015 - .05 ppm); several peaks > 1880 $\mu g/m^3$ (1.0 ppm). Monitoring location not reported. 24-hr avgs by sodium arsenite; peaks by chemiluminescence	128 children 0-5 346 children 6-10 421 children 11-15	No significant difference in reported respiratory illness between homes with gas and electric stoves in children from birth to 12 years	Mitchell et al., 1974 See also Keller et al., 1979a
NO_2 plus other gas stove combustion products	Sample of same households as reported above but no new monitoring reported	174 children under 12	No evidence that cooking mode is associated with the incidence of acute respiratory illness	Keller et al., 1979b
Studies of Adults				
NO_2 plus other gas stove combustion products	Preliminary measurements peak hourly 470 - 940 $\mu g/m^3$ max 1880 $\mu g/m^3$ (1.0 ppm)	Housewives cooking with gas stoves, compared to those cooking with electric stoves	No consistent statistically significant increases in respiratory illness associated with gas stove usage	USEPA, 1976
NO_2 plus other gas stove combustion products	See table above for monitoring	Housewives cooking with gas stoves, compared to those cooking with electric stoves. 146 households	No evidence that cooking with gas associated with an increase in respiratory disease	Keller et al., 1979a
NO_2 plus other gas stove combustion products	See table above for monitoring	Members of 441 households	No significant difference in reported respiratory illness among adults in gas vs electric cooking homes	Mitchell et al., 1974 See also Keller et al., 1979a
NO_2 plus other gas stove combustion products	See table above for monitoring	Members of 120 households (subsample of 441 households above)	No significant difference among adults in acute respiratory disease incidence in gas vs electric cooking homes	Keller et al., 1979b

Two independent sets of epidemiological studies, from Britain and the United States, provide data suggesting likely associations between respiratory illness symptoms in children and residence in homes using gas stoves for cooking versus residence in homes using electrical stoves. Much caution must be employed, however, in interpreting the results of such studies in terms of specifically implicating NO_2 exposures in the etiology of the reported health effects and in attempting to define pertinent exposure/effect relationships.

Results of the British studies have been reported by Melia et al. (1977; 1978; 1979), Goldstein et al. (1979), and Florey et al. (1979). The initial study, conducted from 1973 to 1977, investigated the effects of indoor and outdoor air pollution on respiratory illness in a large cohort of primary school children from randomly selected areas of England and Scotland. Results for the first year (1973) of the study were reported by Melia et al. (1977). Additional results from the last year of the study (1977) and from longitudinal analyses (1973 to 1977) were reported by Melia et al. (1979).

The cross-sectional analysis of 1973 results discussed by Melia et al. (1977), involved 2,554 children from homes with gas stoves and 3,204 from homes with electric stoves and examined the prevalence of bronchitis, cough, colds going to the chest, wheeze, and asthma by means of questionnaires. Crude prevalences for each condition were higher in children from homes where gas was used and statistically significant ($p < 0.05$) for bronchitis, cough, and colds going to the chest in both sexes, and for wheeze in girls. The authors reported that this "cooking effect" appeared to be independent of the effects of age, social class, latitude, population density, family size, overcrowding, outdoor levels of particulate matter (smoke) and sulfur dioxide, and types of fuel used for heating. This conclusion was based on the proportion of children with more than one disease or symptom being higher for homes with gas cooking when these various factors were taken into account; however, when the factors were taken into account, the main finding of the proportion of children with one or more respiratory symptoms or diseases remaining higher in both boys and girls from gas stove homes only approached statistical significance for girls ($p \simeq 0.10$) but not boys. Furthermore, data for several of the variables were missing so that only very small numbers remained within necessary subgroups, prompting the authors to state that the results from these analyses were not conclusive and needed to be confirmed by follow-up data then being collected. Nevertheless, the authors concluded that elevated levels of nitrogen oxides arising from combustion of gas might be the cause of the increased respiratory illness.

In 1977, another cross-sectional study of similar design was conducted on a different set of children, 3,017 from homes with an electric stove, and 1,810 from homes with a gas stove (Melia et al., 1979). Crude prevalences indicated that cough in boys ($p \simeq 0.02$), and colds going to the chest in girls ($p < 0.05$) were significantly higher in homes with gas stoves.

When prevalences of the respiratory conditions were grouped, an association of gas cooking with occurrence of one or more respiratory conditions was found in both sexes ($p \simeq 0.01$ in boys; $p = 0.07$ in girls). When possible confounding or covarying factors considered in 1973, plus smoking among family members, were taken into account, an association between gas cooking and respiratory illness was found in urban areas ($p < 0.005$ in boys, $p \simeq 0.08$ in girls), but not rural ones. In rural areas there only appeared to be an association for girls under eight years old. The effect of number of smokers was significant in rural areas ($p < 0.005$).

In addition to results for the 1977 cross-sectional analysis, Melia et al. (1979) reported that four cohorts of children from the 1973 study who were followed up for 1 to 4 years showed greater risk of having one or more respiratory symptoms or diseases in homes with gas stoves relative to homes with electric stoves in 1973. In later years, however, as the cohorts grew older, the relative risk showed considerable variation. In most groups the risk was greater in gas stove homes, but there were groups with negligible increased risk and groups for which the risk was greater in homes with electric stoves. The authors report no consistent change in the size of the relative risk with time except possibly a decline from 1973 to 1977 for the youngest cohort. Comparing the 1973 and 1977 results, the effect of gas cooking seemed to be smaller in 1977, at least among girls. The prevalences tended to be higher in 1973 for children of the same age. The authors stated that differences in weather conditions could not explain this observation, but speculated that past high levels of atmospheric pollution may have contributed to these results. Nevertheless, Melia et al. (1979) stated, in summary, that they observed an association between respiratory illness and the use of gas for cooking in two different groups of children seen 4 years apart in their national survey.

In another study by Melia et al. (1978) concentrations of NO_2 were determined in the kitchens of two gas cooking homes and two electric cooking homes. Concentrations of NO_2 were determined using TEA diffusion tubes (Palmes, 1976) placed 0.6 m and 2.2 m from the kitchen stove. The average hourly concentration of NO_2 in gas kitchens was 135 $\mu g/m^3$ (0.072 ppm), and in electric kitchens it was 17 $\mu g/m^3$ (0.009 ppm); the difference in these levels was significant at $p < 0.05$. This study also established that reproducibility of the diffusion tubes was ± 3 percent.

These studies by Melia et al. provide suggestive evidence of an association between the use of gas stoves and increased incidence of acute respiratory disease symptoms in children, and between the use of gas stoves and increased levels of NO_2 in the home. However, because NO_2 concentrations were not directly monitored in the homes used in the health studies, only these qualitative conclusions may be drawn regarding their study results.

Possible interrelationships between NO_2 exposures in gas stove homes and increased respiratory infection and decreased lung function were investigated in a further British study reported by Goldstein et al. (1979) and Florey et al. (1979). In these later investigations levels of NO_2 were determined in gas and electric cooking homes, and the prevalence of respiratory symptoms among children in the gas cooking homes was found to be higher than in the electric stove homes ($p \simeq 0.10$). The sample was 808 children aged 6 to 7 years from 769 different homes in a 4-square-km area in Cleveland, UK. The study was conducted for a two-week period during February 1978. NO_2 was measured by TEA diffusion tubes attached to walls in the kitchen area and in the children's bedrooms. In homes with gas stoves, levels of NO_2 in kitchens ranged from 10 to 596 $\mu g/m^3$ (0.005 to 0.317 ppm) with a mean of 211 $\mu g/m^3$ (0.112 ppm), and levels in bedrooms ranged from 8 to 318 $\mu g/m^3$ (0.004 to 0.169 ppm) with a mean of 56 $\mu g/m^3$ (0.031 ppm). In homes with electric stoves, levels of NO_2 in kitchens ranged from 11 to 353 $\mu g/m^3$ (0.006 to 0.188 ppm) with a mean of 34 $\mu g/m^3$ (0.018 ppm), and in bedrooms NO_2 levels ranged from 6 to 70 $\mu g/m^3$ (0.003 to 0.37 ppm) with a mean of 26 $\mu g/m^3$ (0.014 ppm). Outdoor levels of NO_2 were determined using diffusion tubes systematically located throughout the area, and the weekly average ranged from 26 to 45 $\mu g/m^3$ (0.014 to 0.024 ppm).

Information on the prevalence of respiratory symptoms was collected and grouped as in previous studies. Cooking fuel was found to be associated with respiratory illness, independent of social class, age, sex, or presence of a smoker in the house ($p = 0.06$). However, when social class was excluded from the regression, the association was weaker ($p \simeq 0.11$). For the 6- to 7-year-old children living in gas stove homes, there appeared to be an increase of respiratory illness with increasing levels of NO_2 in their bedrooms ($p \simeq 0.10$), but no significant relationship was found between respiratory symptoms in those children or their siblings or parents and levels of NO_2 in kitchens. Lung function tests ($FEV_{0.75}$, PEFR, MMF) were also performed on the 6- to 7-year-old children, but no significant relationship was found between lung function and concentrations of NO_2 in either kitchen or bedroom. These studies, therefore, at most found a weak association between prevalence of respiratory illness in 6- to 7-year-old children and gas cooking in their homes which may have been due to levels of NO_2 generated by the use of gas. The authors note, however, that the NO_2 levels might possibly be a proxy for some other factor more directly related to respiratory disease, such as temperature or humidity.

Results from United States studies finding associations between gas stove use and increased respiratory illness in children have also been published (Spengler et al, 1979; Speizer et al, 1980). Spengler et al. (1979) presented data on annual nitrogen dioxide concentrations inside and outside electric- and gas-cooking homes in five of six American communities included in a prospective epidemiological investigation. In all instances, houses with gas facilities had higher levels of NO_2 than were present in the outdoor air, reaching double the outdoor concentration in some instances. Indoor annual average values in these houses were as high as 80 $\mu g/m^3$ (0.04 ppm). Short-term peak levels in excess of 1,100 $\mu g/m^3$ (0.58 ppm) occurred regularly in kitchens. Houses with electric cooking services had lower concentrations than were observed in the outdoor environment.

Respiratory disease rates were evaluated in these same six communities by Speizer et al. (1980). Children from households with gas stoves had a significantly greater history of serious respiratory illness before age 2 (average difference 32.5/1000 children). In this study, adjustment of rates of illness before age 2 for parental smoking and socioeconimic status led to a clear association with presence of gas stoves ($p < 0.01$). These findings corroborate those reported earlier by Melia et al. (1977), but have more importantly removed the primary objection to the earlier Melia studies, i.e., the role of parental smoking. Speizer et al. (1980) also found small but significantly ($p < 0.01$) lower levels of FEV_1 and FVC, corrected for height, in children from houses with gas stoves. Although these reported pulmonary function changes were statistically significant, the authors noted that the changes are most likely of relatively minimal immediate physiological importance.

Considerable caution should be exercised at this time before fully accepting findings of increased incidence of respiratory disease in children living in gas stove homes and attributing such effects to NO_2 exposure. Such caution is warranted in view of the following :

(1) The findings are based on initial data analyses from long-term prospective studies and assume adequate control for and exclusion of contributions of potential confounding factors (e.g., socio-economic status, humidity, temperature) to the reported associations between gas stove home residence and increased respiratory illness history in young children. Confidence in such findings would be greatly enhanced if they were further confirmed by analyses of data subsequently collected in the studies, analyses that more definitively rule out effects of potentially confounding factors.

(2) The need to define better quantitative exposure/effect or dose-response relationships between peak, 24-hr, weekly or annual average NO_2 exposures and any resulting increases in respiratory disease symptoms in young children residing in homes using gas cooking stoves.

(3) Results from other studies discussed below, which did not find significant associations between increased respiratory illness and residence in gas stove homes.

Several studies, all American, have failed to find evidence of associations between gas stove usage and increased respiratory illnesses. For example, in a U.S. Environmental Protection Agency study (Table 15-6) the incidence of acute respiratory illness among women using gas cook stoves was compared with the incidence among women using electric cook stoves (U.S. Environmental Protection Agency, 1976). Concentrations of NO_2 as high as 940 $\mu g/m^3$ (0.5 ppm) to 1880 $\mu g/m^3$ (1.0 ppm) were found for durations of 1/2 to 1 hour each time the gas stove was used for the preparation of a meal. There was no difference in the incidence of respiratory disease in these women.

Keller et al. (1979a) determined the incidence of respiratory disease in 209 suburban Ohio middle-class families with gas stoves and in 232 similar families with electric stoves. Health data were obtained through biweekly telephone calls for one year. In addition, pulmonary function tests ($FEV_{0.75}$ and FVC) were conducted on a 42 percent sample of the participants representative of both sexes and both types of households. No differences in illness rates or in the results of pulmonary function tests were detected. Nitrogen dioxide and NO levels were monitored over 24-hour periods in 83 of the homes with gas stoves. For this monitoring, a sodium arsenite procedure was used and continuous chemiluminescence measurements were made for 3-day periods in each of 46 homes. Reported peak NO_2 concentrations in homes with gas stoves were as much as 8 times higher than the 24-hour mean and sometimes exceeded 1,880 $\mu g/m^3$ (1.0 ppm). The location of the peak concentrations within the home was not reported, but probably was within a few feet of the stove. On the basis of the range of mean NO_2 concentrations reported, it can be determined that peak (15-min) NO_2 concentrations in most homes with gas stoves ranged between 75 and 1,650 $\mu g/m^3$ (0.04 and 0.88 ppm). The average peak value would have been approximately 750 $\mu g/m^3$ (0.4 ppm). In homes with electric stoves, the mean NO_2 concentration was lower than the mean of 53 outdoor determinations.

Keller et al. (1979b) extended this study of gas versus electric cooking services on 120 of the original households with school age children. Reports of respiratory illness and symptoms were obtained by telephone interview every 2 weeks for 13 months. When the onset of respiratory illness occurred within 3 days of a call, a household visit was made to examine the ill person and to obtain a throat culture. "Well" controls were also examined. The only significant difference ($p < 0.05$) in the incidence of reported acute respiratory illness occurred among children 12 to 18 years of age, with a larger percentage occurring in electric-stove households. Symptoms of "tearing and redness of eye" and frequency of consulting physicians were higher in homes with gas stoves. However, overall, no significant differences were found between the two groups confirming the earlier finding by Keller et al. (1979a) that there was no evidence that gas or electricity in households was associated with the incidence of acute respiratory illness. (A more detailed account of these studies is contained in two reports by Lutz et al., 1974,1977.) Nitrogen dioxide and NO concentrations were monitored over periods of 24 hours using a sodium arsenite procedure and continuous chemiluminescence.

15.3.3 Effects of NO_2 Pollution on Prevalence of Chronic Respiratory Disease

Only a few published epidemiological studies have attempted to investigate possible associations between ambient air exposures to NO_2 or other NO_x compounds and the prevalence of chronic respiratory diseases. The results of such studies are concisely assessed below.

The prevalence of chronic bronchitis among Japanese post office employees in 1962 and in 1967 was investigated by Fujita et al. (1969). Nearly 7,800 employees in Tokyo, Tsurumi, and Kawasaki, Japan were categorized on the basis of their work sites being "downtown and industrial," "intermediate," or "suburban." Chronic bronchitis rates were higher in 1967 than in 1962 for all age groups, for all smoking categories, and for employees who worked both indoors and outdoors. Overall bronchitis rates associated with downtown, intermediate, and suburban areas increased, respectively, from 5.0, 3.7, and 3.7 per 100 employees in 1962 to 8.4, 8.0, and 8.1 per 100 employees in 1967. The investigators speculated that the increases in chronic bronchitis were caused by concomittant increases in the atmospheric concentrations of NO_2, NO, and SO_2, but the aerometric data available for the 1962-67 study period are insufficient to establish any such hypothesized relationships.

Another study of chronic bronchitis among 400 housewives (30 to 39 years old) living in six localities in Japan was reported by the Central Council for Control of Environmental Pollution (1977). This study, conducted during the winter of 1970-71, reportedly found an association between prevalence rates for chronic bronchitis in areas where annual average NO_2 concentrations were in the range of 0.02 to 0.03 ppm. However, insufficient information was provided by which to evaluate the validity of the reported findings, and population NO_2 exposure in this study was likely confounded by high atmospheric particle levels also reported to be present in the same study areas.

In an American study discussed earlier, Speizer and Ferris (1973a) compared the prevalence of chronic respiratory disease among 128 policemen who patrolled on foot in congested business and shopping areas of central Boston with that of 140 suburban patrol car officers. The exposure of each group to NO_2 was determined at several work locations for the central city officers and in the patrol cars of suburban officers. Nitrogen dioxide was measured by the Saltzman method. Among urban policemen, small but not statistically significant increases in the prevalence of chronic respiratory disease were found among nonsmokers and smokers but not among ex-smokers. Estimates of annual mean pollution levels, based on approximately 1,000 hourly samples (Burgess et al., 1973), were, for the urban area, 103 $\mu g/m^3$ (0.055 ppm) NO_2 together with SO_2 concentrations of 90 $\mu g/m^3$ (0.05 ppm); the NO_2 concentrations for the suburban area averaged 75 $\mu g/m^3$ (0.04 ppm) and SO_2 concentrations averaged 26 $\mu g/m^3$ (0.01 ppm).

Cohen et al. (1972) similarly found no differences in the prevalence of chronic respiratory disease between a nonsmoking population in the Los Angeles basin and a similar population in San Diego. The Los Angeles group was exposed to concentrations of NO_2 between 90 and 100 $\mu g/m^3$ (0.05 ppm) plus oxidant levels of about 90 $\mu g/m^3$ (0.045 ppm); the San Diego group was exposed to NO_2 concentrations of approximately 40 to 45 $\mu g/m^3$ (0.02 ppm) and concentrations of oxidants of approximately 76 $\mu g/m^3$ (0.038 ppm).

Linn et al. (1976) also found no increase in chronic respiratory disease in Los Angeles women exposed to a median hourly NO_2 concentration of 130 $\mu g/m^3$ (0.07 ppm) with a 90th percentile value of 250 $\mu g/m^3$ (0.13 ppm), over that occurring in San Francisco women where the median hourly concentration of NO_2 was 65 $\mu g/m^3$ (0.035 ppm) and the 90th percentile was 110 $\mu g/m^3$ (0.06 ppm). Median hourly oxidant values in the two areas were, respectively, 0.07 and 0.02 ppm. These investigators concluded that cigarette smoking was much more significant than was Los Angeles air pollution in the development of chronic respiratory illness.

In summary, the epidemiology studies assessed above did not establish any credible association between chronic respiratory disease prevalence in human populations and the concentrations of NO_2 to which these populations were exposed.

15.3.4 Extrapulmonary Effects of Exposure to NO_x

Nitrogen oxides, as well as other components of polluted air, have been reported to be correlates of daily mortality, heart disease, and lung cancer, based on certain epidemiologic studies reported in the early 1970's.

For example, Hickey et al. (1970) compared air pollution measurements made at National Air Sampling Network (NASN) stations with geographical differences in mortality rates for various categories of cancer, cardiovascular disease, and respiratory disease in 38 U.S. Standard Metropolitan Statistical Areas (SMSAs) during 1959 to 1961 and from 1961 to 1964. Included in the analyses were NO_2, SO_2, suspended sulfates, total particulates, calcium, chromium, copper, iron, lead, manganese, nickel, tin, titanium, vanadium, zinc, and water hardness. Mortality rates were analyzed both with and without regard to age, sex, and race differences. Nitrogen dioxide and SO_2 repeatedly were positively associated with age-, race-, and sex-adjusted, and unadjusted mortality rates for various cancers, and for arteriosclerotic heart disease. Other pollutants were variably, and often negatively, associated with these mortality categories. However, the quality of both the monitoring data and the mortality data, as well as the fact that specific pollutant exposures of individuals dying of these different diseases could not be evaluated, are such that the findings preclude more than speculative conclusions regarding possible risk of death or disease due to pollutant exposure.

Lebowitz (1971) studied variations in daily mortality in relation to daily air pollution and weather variables in New York City, Philadelphia and St. Louis, from 1962 to 1965 and reported associations between air pollution, weather variables, and daily mortality for each city. Multiple regression analyses showed a significant negative association between winter mortality in New York City and daily nitrogen oxide concentrations (non-specific for NO, NO_2 or other NO_x), but no association in summer. In contrast, the winter mortality of persons 45 to 64 years old, 65 years and older, and all ages combined in Los Angeles, California, was significantly and positively related to daily nitrogen oxide concentrations during the period of 1962 to 1969 but summer mortality was not. These results, therefore, do not provide convincing evidence of a relation between NO_x air pollution and daily mortality, given that winter mortality results for New York City were opposite to those for Los Angeles and the associations were not consistent across seasons.

Interactions of atmospheric NO_x pathways with those of photochemical oxidants, discussed elsewhere in this document and also in the Air Quality Criteria for Ozone and Other Photochemical Oxidants, (U.S. Environmental Protection Agency, 1978) may lead to increases in the incidence of skin cancer in certain population groups. Epidemiological studies have demonstrated that solar UV radiation is carcinogenic (Douglas and Owen, 1976). Outdoor workers, such as farmers or fishermen, have a higher incidence of both basal cell and squamous cell carcinoma of the skin than do less-exposed individuals. Chemical reactions in the atmosphere involving nitrogenous compounds may lead to a decrease in the stratospheric concentration of O_3 and a resultant increase in the amount of UV radiation penetrating to the earth's surface, but such effects have not yet been conclusively demonstrated.

15.4 ACCIDENTAL AND OCCUPATIONAL EXPOSURES

As noted earlier in this chapter, there are a number of occupational situations in which workers are intermittently or continuously exposed to high concentrations of NO_2 or other oxides of nitrogen. Data from such exposures or other accidental exposures, as those encountered with certain fires, provide some indications of NO_x exposure levels associated with severe toxic effects in humans.

There have been a few cases of unusually high levels of exposure for short periods of time which confirm a potential lethal hazard associated with short-term exposure to NO_2. Lowry and Schuman (1956) reported the development of illness of four farmers who entered freshly-filled silos in which high concentrations of NO_2 had built up. These men experienced cough and dyspnea shortly after entering the silos. These symptoms disappeared after several days, but were followed in about 3 weeks by cough, malaise, weakness, dyspnea, and fever. Chest X-rays showed multiple discrete nodules scattered in both lungs. Two of the patients died while the other two improved dramatically after receiving high doses of steroids.

Concentrations of NO_2 were estimated to be in the range of 380,000 to 7,500,000 μg/m^3 (200 to 4,000 ppm).

Grayson (1956) reported on two other cases of NO_2 poisoning from silage gas estimated at 560,000 to 940,000 μg/m^3 (300 to 500 ppm) NO_2. Indications from this study are that exposure to concentrations in this range is likely to result in fatal pulmonary edema or asphyxia. The study further indicated that concentrations in the range of 280,000 to 380,000 μg/m^3 (150 to 200 ppm) are likely to produce bronchiolitis; exposure to 94,000 to 190,000 μg/m^3 (50 to 100 ppm) are associated with reversible bronchiolitis, and exposure to concentrations in the range of 47,000 to 140,000 μg/m^3 (25 to 75 ppm) are associated with bronchitis or bronchial pneumonia with apparent complete recovery probable.

Gregory et al. (1969) studied mortality of survivors of a fire at Cleveland Clinic (Cleveland, Ohio), in May 1929. At the time of the fire, persons were exposed to high concentrations of NO, NO_2, CO, and hydrogen cyanide resulting from the combustion of X-ray film in which nitrocellulose was a basic material. Exposure was such that it caused 97 deaths within 2 hours and, over the next 30 days, 26 died. Under such extreme conditions, several factors, including various atmospheric pollutants, may have contributed to the immediate deaths. The conditions at the time of the fire, however, and the symptoms in many of the individuals who subsequently died, were most consistent with symptoms expected as a result of inhalation of very high NO_2 concentrations. In spite of the significant number of deaths within 30 days of the fire, the survival rate over the next 30 years for exposed clinic employees, firemen, policemen, and rescue workers did not differ from that of unexposed similar groups. This suggested an absence of residual effects (excess mortality) due to the intense acute exposure.

Another study (Lowry and Schuman, 1956), performed on 70 male chemical workers exposed to 750 to 5000 μg/m^3 (0.4 to 2.7 ppm) NO_2 in their work place daily for 4-6 years, compared various blood lipid concentrations in these subjects to values obtained on a control group of 80 men not exposed to NO_2. They reported that lipid metabolism was impaired subsequent to the original NO_2 exposure. Adequate information to evaluate the significance of this finding was not available. However, as discussed earlier, Horvath et al (1978) reviewed case studies for acute NO_2 exposure victims which also suggest that certain pulmonary function decrements may persist for periods up to 13 years after initial exposure.

15.5 EFFECTS OF NO_x-DERIVED COMPOUNDS

Many compounds may be derived from various oxides of nitrogen in the atmosphere, with formation mechanisms and concentrations depending on many factors including the concentration of various nitrogen and non-nitrogen materials present, temperature, humidity, and sunlight. The compounds believed to represent the greatest potential risk to health include nitric acid, nitrates, nitrites, and nitrosamines.

15.5.1 Nitrates, Nitrites and Nitric Acid

Nitrate poisoning occurs when a sufficient quantity of nitrate ions is reduced by intestinal bacteria to nitrites, which, in turn, oxidize the iron in hemoglobin from the ferrous to the ferric state. The resulting substance, termed methemoglobin, cannot function normally in the process of transporting oxygen to tissues. In healthy adults, methemoglobin usually accounts for less than 2.0 percent of the total hemoglobin concentration (National Academy of Sciences, 1972). However, Goldsmith et al. (1975) reported results of a study of California populations in which the mean concentrations in populations ranged as high as 2.11 percent methemoglobin, with 1 percent of adults and 8 percent of infants exceeding 4.0 percent methemoglobin. Infants usually carry higher concentrations of methemoglobin and are more susceptible to nitrate poisoning than are older children or adults because (1) fetal hemoglobin is probably more susceptible to conversion to methemoglobin, (2) bacteria capable of reducing nitrate to nitrite thrive in the less acidic conditions of the infant stomach, (3) the enzyme system for reducing methemoglobin to hemoglobin is deficient in infants, and (4) because intake of water per kilogram body weight is higher in the infant than in adults (Kravitz et al., 1956). Cyanosis may be produced at concentrations of about 10 percent methemoglobin; however, symptoms are not likely to become obvious at concentrations less than 20 percent.

The total weekly intake of nitrate in the general populations of the United States (Ashton, 1970) and in England (Hill et al., 1973) has been estimated to average about 400,000 to 500,000 μg. Because concentrations in water, in cured meats, and in vegetables vary greatly, as do the quantities of these materials consumed by individuals, the ingestion estimates must be applied with caution. However, since the worst case situation would probably find less than 40 $\mu g/m^3$ nitrate in ambient air (Pitts and Loyd, 1973), an adult engaged in heavy exercise, who might inhale 20 m^3 of air per day could be expected to inhale no more than 5,600 μg of nitrate per week, or less than 1.5 percent of the lowest estimate of total weekly intake. Thus, it is considered to be unlikely that the concentrations of nitrate in the ambient air contribute significantly to the production of acute nitrate poisoning.

Nitrate aerosols could be significant from an air pollution standpoint in that they are the final stage in the atmospheric oxidation of NO_x, a process that includes the formation of various nitrogenous acids. However, the earlier discussion of measurement techniques (Section 7.4.1) suggest that many of the data now available relevant to atmospheric concentrations of nitrates represent measurements of the atmospheric nitrate plus artifact nitrate formed on the collection filter by the reaction between the filter substrate and nitrogen compounds including nitric acid. No published epidemiological studies on the effects of atmospheric nitrates are yet available where the ambient air levels of nitrates were measured following collection of particulate matter glass filters (which substantially avoids artifact formation).

Only a few recent controlled human exposure studies have attempted to assess the effects of inhaled nitrates on pulmonary functions, using acceptable nitrate measurement methods. For example, Utell et al. (1979) studied the effects of a nitrate aerosol on pulmonary function and on the sensitivity of test subjects to the bronchoconstricting effects of carbachol. Included in these studies were 7 healthy subjects (mean age 28) and 13 mild asthmatics (mean age 25) selected on the basis of a demonstrated abnormal increase in R_{aw} after inhaling carbachol. Subjects were exposed for 16 minutes in a double-blind manner to either sodium nitrate ($NaNO_3$) or sodium chloride (aerodynamic diameters 0.49 μm, $\sigma g = 1.7$, and 0.46 μm, $\sigma g = 1.7$, respectively) at a concentration of 7,000 μg/m^3. Following exposure, subjects inhaled a predetermined quantity of carbachol sufficient to increase R_{aw} 20 to 30 percent. Prior to exposure, after 8 and 16 minutes of exposure, and again after the inhalation of carbachol, the following pulmonary measurements were made: functional residual capacity (FRC), R_{aw}, FEV, $FEV_{1.0}$, maximum and partial expiratory flow rates at 60 and 40 percent total lung capacity. None of the tests of pulmonary function were affected by the nitrate exposure although two of the asthmatic subjects did demonstrate mild potentiation of the response to carbachol after nitrate exposure. All subjects remained asymptomatic. These results suggested that in healthy individuals or in mild asthmatics, short-term exposure to $NaNO_3$ at concentrations approximately 100 times the total nitrate in ambient air exposures, does not affect pulmonary function. These investigators did point out that their study results might have been quite different had they (1) used an "acidic" nitrate in their exposure atmosphere rather than the neutral $NaNO_3$, (2) had the exposure time been extended beyond the 16 minutes, or (3) had the study included symptomatic asthmatics.

Utell et al. (1980) also examined the potential synergy between acute exposure to a pollutant (sodium nitrate aerosol) and acute respiratory infections. The mass median aerodynamic diameter of the aerosol was 0.49 μm; the concentration 7,000 μg/m^3. Eleven previously healthy adults with uncomplicated influenza A (H_1N_1) were studied at the time of acute illness and 1, 3, and 6 weeks later. Significant decreases in specific airway conductance and partial expiratory flows at 40% of total lung capacity were observed at the initial examination and 1 week later. By the third week, inhalation of sodium nitrate no longer produced changes in air way function. Control studies were made with sodium chloride aerosol. They concluded that individuals with acute respiratory disease were susceptible to bronchoconstriction from this air pollutant--one that normally did not influence airway function.

"Nitric acid fumes," a term used to designate the mixture of nitric acid vapor plus the reaction products of nitric acid and various metals or organic material, has been known to produce varying degrees of upper respiratory irritation within minutes of exposure. Prognosis for exposed individuals depends upon the concentration of the acid plus its products of reaction and the duration of exposure. The clinical picture sometimes is biphasic and similar to that shown by individuals exposed to high concentrations of NO_2 (Treiger and Przypyszny, 1947). In other instances, the picture is quite different and may reflect the toxicity of reaction products, particularly those produced by the reaction of nitric acid and some metals (Danke and Warrack, 1958). Extended exposure to lower concentrations of nitric acid vapors have been postulated as the probable cause of chronic bronchitis or a chemical pneumonitis (Fairhall, 1957). Neither these effects nor the concentration that might cause them are well documented.

There are no data available relating to the effects of inhaling nitric acid vapors in concentrations likely to occur in the ambient atmosphere; however, it does seem likely that such a highly ionized and strongly corrosive material would be a potent respiratory irritant even at low concentrations.

15.5.2 Nitrosamines

A few epidemiological studies have attempted to link environmental nitrates, nitrites, and nitroso compounds with human cancer. The International Agency for Research on Cancer (IARC) investigated a possible association between these compounds in the diet and esophageal cancer in specific areas of Iran and France, where these tumors occur at a high rate and in nearby areas where the tumor rates are lower (Bogovski, 1974). Fifteen of 29 samples of cider contained 1 to 10 μg/kg of dimethylnitrosamine and two samples also contained diethylnitrosamine (1 μg/kg). Benzo(a)pyrene also occurred in some samples. Correlations between dietary intake of N-nitroso compounds and esophageal cancer were not established (Bogovski, 1974).

A similar study was conducted in the Anyang region of China, where 20 percent of deaths from all causes reportedly result from esophageal cancer (Cordination Group for Research on Etiology of Esophagal Cancer in North China, 1975). Twenty-three percent of the food samples from the areas with the highest cancer rates were reported to contain dimethyl-, diethyl-, and methylbenzyl-nitrosamine. Confirmation of this analysis by gas chromatography and mass spectroscopy, however, is required before the finding can be accepted. Dietary nitrite levels were higher in the areas of high cancer incidence than in low incidence areas. Chickens in the area associated with high esophageal cancer in humans also had a high incidence of similar tumors, suggesting an environmental etiology for the disease.

Zaldivar and Wetterstand (1975) and Armijo and Coulson (1975) have shown some correlation between the per capita use of fertilizer and the incidence of stomach cancer in Chile. It has been hypothesized that nitrate from fertilizer first enters the diet by way of meat, vegetables, and drinking water. Nitrates are then reduced to nitrites by microbial action, and are thus available for *in vivo* nitrosation of secondary amines, contained in the diet, to form carcinogenic nitrosamines, which can induce stomach cancer. The suggested causal relationship remains highly speculative. Hill et al. (1973) correlated variations in rates of stomach cancer with the nitrate content of drinking water in two English towns. However, the evidence required to demonstrate a causal role for nitrate was not provided. Gelperin et al. (1976) found no statistically significant differences in death rates from cancers of several organs, in three areas of Illinois each with different nitrate content in the drinking water. It is doubtful, however, that the available mortality data permitted an analysis that could have detected an effect among the populations in the high-nitrate area. In Japan, increased rates of stomach cancer have been observed in population groups having unusually high consumption of salt-preserved foods (Sato et al., 1959). Reference is made to Chapter 8 for a review of observed concentrations of N-nitroso compounds in ambient air. The relative significance of the inhalation of the compounds is unknown.

There is no direct evidence that atmospheric nitrogenous compounds contribute significantly to the in vivo formation of nitrosamines in humans or that inhaled nitrosamines represent significant health hazards. Questions have been raised as to in vivo nitrosation by NO_2. Iqbal et al. (1980) have reported that, indeed, in vivo nitrosation of amines does occur in mice. There are a number of endogenous and exogenous amines available to an organism for nitrosation. In the preliminary studies of Iqbal et al., they gavaged mice with an exogenous amine, morpholine, and then exposed mice for 0.5 hour to 50 ppm NO_2. Production of N-nitrosomorpholine (NMOR) was measured in the mouse. Longer exposures to this concentration of NO_2 for up to 4 hours resulted in higher levels of the nitrosamine. They also exposed gavaged mice for 4 hours to NO_2 (from 0.2 to 50 ppm) and reported increased levels of NMOR. The site or mechanisms of the NMOR biosynthesis were not identified.

Nitrosation of amines in the stomach has been demonstrated to occur in humans (Sander and Seif, 1969), in rodents (Sander et al., 1968), and in dogs (Mysliwy et al., 1974). Preformed nitrosamines have been found in tobacco (Hecht et al., 1974; Hoffman et al., 1974) and in tobacco smoke (Klus and Kuhn, 1973; Neurath, 1967).

15.5.3 Other Compounds

Recent studies by Pitts et al. (1978) have demonstrated that, in sunlight, very low concentrations of diethylamine and triethylamines behaved like hydrocarbons and reacted with NO, NO_2 or nitrous acid to form O_3, PAN, acetaldehyde plus diethylnitramine and several amides including acetamide. Peroxyacetyl nitrate (PAN) is a strong eye irritant at a concentration of 4,945 $\mu g/m^3$ (1.0 ppm). The effects of this compound are discussed at length in Air Quality Criteria for Ozone and Other Photochemical Oxidants (U.S. Environmental Protection Agency, 1978). In addition to nitrosamines, diethylnitramine (Goodall and Kennedy, 1976; Druckey et al., 1961) and acetamide (Jackson and Dessau, 1961; Weisberger, et al. 1969) have been shown to be carcinogenic in test animals. However, the significance of these materials as human carcinogens is unknown.

15.6 SUMMARY AND CONCLUSIONS

Critical NO_x human health effects issues include: (1) qualitative characterization of identifiable health effects associated with exposure to various NO_x compounds; (2) delineation of the seriousness of the identified effects in terms of their reversibility/irreversibility and their impact on normal human functions and activities; (3) quantitative characterization of exposure/effect or exposure/response relationships for health effects of concern; and (4) identification of sensitive populations at special risk for manifestation of such effects at ambient air NO_2 levels.

Of the oxides of nitrogen, NO_2 appears to be the compound of greatest concern in terms of human health effects documented as likely to occur at exposure levels within the range of or approaching ambient air concentrations encountered in the United States. Based on this, the main focus of the present section is on the summarization of key points and conclusions regarding NO_2 health effects and their likely significance for protection of public health. Brief comments are also provided in regard to certain NO_x-derived compounds hypothesized to be of public health concern.

15.6.1 NO_2 Effects

A broad spectrum of human and animal health effects have been reported to be associated with NO_2 exposure. These range from (1) death or serious, irreversible lung damage associated with very high accidental or experimental exposures in the range of 150-300 ppm or higher; through (2) less severe but clearly significant short-term or chronic lung tissue damage, functional impairment, or aggravation of other disease processes at exposure levels of 5 to 100 ppm; to (3) impairment of lung defense mechanisms and other, milder, temporary effects, e.g.,changes in pulmonary function and sensory system effects, occurring at NO_2 levels below 5 ppm. Of most relevance here is consideration of the effects occurring at levels below 5 ppm.

15.6.1.1 Pulmonary Function Effects--Numerous controlled human exposure studies have examined the effects of single, short-term NO_2 exposures on pulmonary function. The most frequently observed pulmonary function effects of NO_2 exposure in controlled human studies (usually at concentrations higher than ambient) include increases in airway resistance (R_{aw}) and changes in susceptibility to the effects of bronchoconstricting agents. Functional response of human subjects to NO_2 are commonly assessed with measurements of flow resistance (total respiratory, pulmonary, or airway) and maximum expiratory flow rate (related to lung volume). Both types of measurements are influenced by changes in diameter of the laryngotracheobronchial system. Airway narrowing, or bronchoconstriction, increases flow resistance and reduces maximal expiratory flow rate.

Increased airway resistance (R_{aw}) and other physiological changes suggesting impaired pulmonary function have been clearly demonstrated to occur in healthy adults with single 2-hr NO_2 exposures ranging from 3760 to 13,200 $\mu g/m^3$ (2.5 to 7.0 ppm). Certain studies also indicate that significant effects occur in healthy subjects with shorter (5-15 min) exposures to the same or possibly lower levels of NO_2 administered either alone or in combination with NaCl aerosol. More specifically, in regard to the latter point, Suzuki and Ishikawa (1965) observed altered respiratory function after exposure of healthy subjects to NO_2 levels of 1300 to 3760 $\mu g/m^3$ (0.7 to 2.0 ppm) for 10 minutes. Their data however, preclude a clear association of observed effects with any particular concentration in the range of 1300 to 3760 $\mu g/m^3$ (0.7 to 2.0 ppm) NO_2 exposure.

In contrast, Hackney, et al. (1978) reported no statistically significant changes in any of the pulmonary functions tested with the exception of a marginal loss in forced vital capacity after exposure to 1880 $\mu g/m^3$ (1.0 ppm) NO_2 for 2 hours on two successive days (1.5% mean decrease, $P < 0.05$). However, the authors question the health significance of this small, but statistically significant change and suggest that it may simply be due to random variation. Also, Beil and Ulmer (1976) and Folinsbee et al. (1978) concluded that there were no physiologically significant pulmonary effects at exposure levels of 1880 and 1100 $\mu g/m^3$ (1.0 and 0.6 ppm) NO_2, respectively; and in a similar study, where Kerr et al. (1979) exposed 10 healthy adults to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 2 hours, only small changes were found in quasistatic compliance. However, the authors again suggested that such results may be due to chance alone, especially since no other pulmonary function tests showed significant changes for the healthy adult group and only one subject reported mild symptomatic effects associated with NO_2 exposure. In general, then, these studies appear to have found no observed effect levels for pulmonary function changes in healthy adults across a range of 0.5 to 1.0 ppm NO_2.

Hackney et al. (1975) and von Nieding et al. (1977) also concluded that there were no physiologically significant effects at NO_2 levels below 560 $\mu g/m^3$ (0.3 ppm) in the presence of various other air pollutants, with the possible exception of increased sensitivity to a bronchoconstrictor (acetylcholine) observed by von Nieding et al. (1977) at 94 $\mu g/m^3$ (0.05 ppm) NO_2 in the presence of 49 $\mu g/m^3$ (0.025 ppm) ozone and 290 $\mu g/m^3$ (0.11 ppm) SO_2. The von Nieding finding, however, is difficult to interpret in view of: (1) controversy over the health significance of altered sensitivity to bronchoconstrictors in healthy subjects; (2) uncertainties due to methodological differences between his techniques and other investigators'; and (3) no confirmation of von Nieding's et al. (1977) findings by other investigators. Thus, no presently available controlled human exposure study can be accepted at this time as providing conclusive evidence for meaningful pulmonary function changes occurring at NO_2 levels below 1880 $\mu g/m^3$ (1.0 ppm) for healthy adult subjects.

It is difficult to assess the health significance of small reductions in pulmonary function, eg. increased R_{aw}, reported for healthy adults with exposures to NO_2 below 2.0 ppm; nor have controlled exposure studies investigated long-term effects of repeated NO_2 exposures. Secondary effects of brochoconstriction can add to the biological significance of observed functional alterations. One such possible secondary effect is the interference with alveolar-capillary gas exchange (measured as an increase in alveolar-arterial PO_2 gradient, or as a reduction in arterial PO_2 or oxyhemoglobin saturation). The relation, if any, between bronchoconstriction associated with a single exposure to NO_2 and possible development of chronic respiratory disease during prolonged or intermittent exposure is unknown. It is unlikely, however, that the slight increases in airway resistance which were observed to occur after a single exposure to NO_2 at or below 2.0 ppm represent a significant adverse health effect for healthy adults.

More difficult to interpret is the health significance of NO_2 exacerbation of pulmonary function changes induced by bronchoconstrictors. Monitoring a subject's response to a bronchoconstrictor is a sensitive experimental approach that utilizes (1) the action of neurotransmitters (e.g., acetylcholine) normally present in the body, (2) pharmacologic products with similar properties (e.g., carbachol), or (3) nervous system-mediated reflexes. The purpose of the bronchoconstrictor is to test the response of the individual's airways to a standardized challenge. This response in individual subjects may be altered by underlying disease such as asthma, by respiratory infection or by previous exposure to an air pollutant. A reasonable hypothesis is that the magnitude of response to a particular experimental challenge such as a synthetic bronchoconstrictor may be used to predict the individual's level of risk when exposed to ambient pollutants. However, it is also known that increases in bronchial sensitivity can be produced by sudden changes in temperature or even by emotional stress. Thus, it is believed to be unlikely that the relatively mild and inconsistent bronchoconstriction produced by a single short exposure to NO_2 represents a health hazard with or without the added increase in constrictive potential due to any of these other natural or induced influences.

Increases in R_{aw} and increased sensitivity to bronchoconstrictors are both suggestive of irritation in the laryngo-tracheo-bronchial system. The consequence of repeated short-term exposures has not been determined in human volunteers; however, studies with animals suggest that repeated exposures to NO_2 levels at or below those associated with significant increases in R_{aw} can increase susceptibility to respiratory infections.

For purposes of this review, the intensity of the response in test individuals has been given little consideration in assessing potential health risk. This approach was taken because the intent, usually, was to determine the lowest NO_2 concentration that would cause

any increase in R_{aw} in healthy or in susceptible individuals after a single, short-term exposure. Obviously, a barely detectable functional response has less serious implications than one associated with disability or distress.

An assessment of the importance of studies showing associations between exposure to NO_2 and increased airway resistance must consider also the known wide variations in susceptibility within human populations. Thus, an exposure sufficient to produce slightly increased airway resistance in healthy individuals may produce much greater and more severe responses in highly susceptible segments of the population, e.g., in those with symptoms of chronic respiratory illness or asthmatics.

Several controlled clinical studies have specifically addressed the issue of whether detectable respiratory effects can be induced by NO_2 in sensitive human subjects at exposure levels below those affecting healthy human adults. For example, the studies by von Nieding et al. (1971; 1973) show that, in persons with chronic bronchitis, concentrations of 7,500 μg and 9,400 μg/m^3 (4.0 and 5.0 ppm) produced decreases in arterial partial pressure of oxygen and increases in the difference between alveolar and arterial partial pressure of oxygen. Exposures to concentrations of NO_2 above 2,800 μg/m^3 (1.5 ppm), for periods considerably less than 1 hour, also produced significant increases in airway resistance. Thus, results for bronchitic individuals and healthy individuals appear to differ little and provide no particular support for the hypothesis that chronic bronchitics are more sensitive to NO_2 exposure than healthy adults.

In another study (Kerr et al., 1979), measurements of pulmonary function were not altered in 13 asthmatics or 7 bronchitics as a result of 2 hours of exposure to 940 μg/m^3 (0.5 ppm) NO_2 when the groups were analyzed separately. When the data for the two groups were analyzed together, however, small but statistically significant changes in quasistatic compliance and functional residual capacity were reported. Nevertheless, the authors state that the changes reported may be due to chance alone. Seven asthmatics and one bronchitic reported some chest discomfort, dyspnea, headache, and/or slight nasal discharge.

In contrast to the above results, exposures to 190 μg/m^3 (0.1 ppm) NO_2 for 1 hour were reported by Orehek et al., (1976) to increase mean airway resistance (R_{aw}) in 13 of 20 asthmatics and to increase the sensitivity to a bronchoconstrictor (carbachol) in these same individuals. However, considerable controversy exists regarding interpretation of the Orehek (1976) study and the health significance of the increased response to a bronchoconstrictor observed in the study. Also, its basic findings remain to be independently replicated.

Based on the above results, conclusive statements regarding the possible special risk status of asthmatics in response to NO_2 exposure cannot be made at this time. The Kerr et al. (1979) study results do suggest, however, that increased occurrence of personal discomfort and symptoms may occur in asthmatics as the result of short-term (2 hr) exposures to 0.5 ppm NO_2.

The above clinical studies provide important data concerning the effects of single short-term NO_2 exposures on healthy young adults and certain groups defined a priori as "sensitive", i.e., bronchitics and asthmatics. However, members of other presumed sensitive populations, e.g., children, the elderly, and individuals with chronic cardiovascular disease, have not been tested in controlled clinical studies and are not likely to be tested in the future because of medical ethics limitations on the use of such subjects in experimental studies. Therefore, statements on whether such individuals are at greater risk than healthy young adults for experiencing respiratory effects with single short-term NO_2 exposures cannot be made based on controlled human exposure studies.

It is informative to compare the above controlled human exposure study results with findings from epidemiological studies investigating possible effects of ambient air NO_2 exposures on pulmonary function parameters in human adults and children. In that regard, no evidence of pulmonary function decrements was obtained in several epidemiological studies (Cohen et al., 1972; Speizer and Ferris 1973a,b; Burgess et al., 1973; Linn et al., 1976) of adults exposed to ambient maximum hourly NO_2 levels as high as 0.50 ppm or annual average NO_2 levels up to 0.06 ppm. These results appear to be consistent with no observed effects for pulmonary function changes in healthy adults generally obtained in the above controlled human exposure studies with exposures to NO_2 levels of 1.0 ppm or below. None of the presently available epidemiological studies, however, provide information regarding NO_2 effects on pulmonary functions in bronchitis or asthmatics. Also, critical evaluation of other epidemiological studies, on children, reveals that such studies to date do not provide useful quantitative information upon which to base estimates of ambient NO_2 levels associated with pulmonary function decrements in children.

15.6.1.2 Acute Respiratory Disease Effects--Epidemiological studies of outdoor and indoor air pollution have also been employed in an effort to demonstrate an association between NO_2 exposures and the occurrence of acute respiratory disease symptoms and illnesses in both human adults and children. Such studies only provide relatively limited evidence for such associations possibly existing at ambient air NO_2 levels.

Only a few American community air pollution studies on the effects of NO_2 on acute respiratory disease are available, but all were found to be of questionable validity due to the use of the Jacobs-Hochheiser technique in measuring atmospheric concentrations of NO_2.

A few reports on other outdoor air pollution studies on the subject were found in the foreign literature, but were such as to preclude confident evaluation of the methodology involved or contained information which precluded reliable attribution of observed health effects to ambient air NO_2 exposures.

Certain other epidemiological studies on indoor air pollution, in contrast to the outdoor pollution studies, appear to provide useful information regarding associations between NO_2 exposures and acute respiratory disease effects. More specifically, some support for accepting the hypothesis that children are at special risk from NO_2-induced increases in acute respiratory illnesses might be derived from certain British and American studies on indoor pollution effects summarized above in Table 15-6.

The British studies by Melia et al. (1977, 1979) provided initial evidence suggesting that increased acute respiratory symptoms and illness rates may occur among school children living in homes using gas stoves for cooking in comparison to children from homes using electric ranges. High temperature gas combustion is a source of NO_2. However, the first study (Melia, 1977) did not take parental smoking into account, and, when socioeconomic status was controlled for in the statistical analyses for each of the two studies, only relatively weak associations ($p \cong .10$) were found for some subgroups of children but not others. Also, NO_2 levels were not monitored in the study homes. In later British studies, by Florey et al. (1979) and Goldstein et al. (1979), NO_2 levels were measured in the kitchens and bedrooms of children residing in gas stove homes; but no significant relationships were found between weekly NO_2 levels in the kitchen and acute respiratory illness symptoms in 6 to 7 year old children or their siblings or parents. Nor were any significant associations found between NO_2 levels and pulmonary function measures in the 6 to 7 year old children. Only some weak associations ($p \cong .10$) were found between increased respiratory illness in the same children and NO_2 levels in their bedrooms. The authors suggest, however, that the apparent association may be due to NO_2 serving as a proxy for some other variable (humidity, temperature) more important in the etiology of acute respiratory infections in the children. The later British studies,therefore, do not appear to have provided convincing evidence to substantiate the initial hypothesis of possible NO_2 -induced increases in acute respiratory disease effects in children residing in gas-stove homes. Consistent with this, United States studies by Keller et al. (1979 a,b) failed to find any evidence supporting the existence of the hypothesized relationship between NO_2 and acute respiratory illness in school-aged American children living in gas stove homes.

On the other hand in the United States study recently reported by Speizer et al. (1980), children from households with gas stoves were reported to have a greater history of serious respiratory illness before age 2. In this study, adjustment of rates of illness before age 2 for parental smoking and socioeconomic status led to a clearly significant ($p < .01$) association with the presence of gas-cooking devices. Also found were small but statistically significant lower levels of two measures of pulmonary function, corrected for height, in children from houses with gas stoves. In all instances in the subset of study homes with gas facilities where NO_2 levels were monitored (Spengler et al., 1979), NO_2 was present in higher concentrations than was present in the outside air. Indoor values monitored in one gas stove home averaged as high as 54 µg/m^3 (0.03 ppm) over a two-week period; and short-term (lasting minutes to hours) NO_2 levels in excess of 500 µg/m^3 (.25 ppm) and even 1,000 µg/m^3 (0.5 ppm) occurred in the kitchen during cooking.

The Speizer/Spengler findings tend to support the original hypotheses initially advanced by Melia et al. (1977) regarding associations between exposure to NO_2 produced by gas combustion and increased acute respiratory disease in children residing in homes using gas stoves. Also, the small but significant decrements in pulmonary function observed by Speizer et al. (1980) in the children at age 6 to 7, while not likely of much immediate physiological or health consequence, may be indicative of persisting residual effects of the early childhood respiratory infections on lung development and growth. Furthermore, other studies by Colley et al. (1973), Kiernan et al. (1976), and Tausseg (1977) suggest that increased rates of respiratory illness in young pre-school children may result in increased susceptability for respiratory infection lasting into early adulthood. Such considerations regarding possible long-term health consequences, together with the obvious immediate health significance of respiratory infections in young children, argue for treating any such effects as being sufficiently adverse as to be of clear concern in terms of public health protection.

Certain factors as noted earlier (page 15-36), however, should be taken into account before fully accepting at this time the above findings and their potential implications. This includes an apparent inconsistency between the Speizer findings and the results of the Keller studies, which found no association between residence in gas-stove usage homes and respiratory illness rates in American school children. Several explanations for the apparent discrepancy can be advanced. For example, the sample sizes for children used in the Keller studies were approximately 10 times smaller than the sample size employed in the Speizer study, thereby reducing the likelihood of Keller demonstrating statistically significant associations between gas stove usage and increased respiratory illness among children from gas stove homes. Also, the associations reported by Speizer were for increased respiratory illness rates before the age of two, whereas the Keller findings apply to older, school-aged children 6 to 7 years old. If both sets of findings are accurate, however, then they may suggest that the increased respiratory infections observed before the age of two by Speizer are not associated, after all, with long-term, persisting consequences of the type hinted at by the Colley and Kiernan findings (vida supra).

If one does accept the Speizer findings as being indicative of increased respiratory infections occurring in young children living in gas stove homes, then the question remains as to what NO_2 exposure levels and durations might be associated with the induction of such effects. Unfortunately, no clear resolution of this issue is presently available. The authors themselves (Speizer et al., 1980) speculate that repeated exposures to intermittently high peak levels of NO_2 may be most important in contributing to the increased incidence of respiratory illness. This hypothesis is based primarily on their observations (Spengler et al., 1979) of long-term NO_2 annual averages levels in gas stove homes not differing much from those found in electric homes, and of the latter lacking the occurrence of intermittent marked 1- or 2-hr indoor air NO_2 peak concentrations associated with periodic use of the gas stoves for cooking.

Estimates of specific NO_2 levels, either for 1-2 hr peaks or for long-term (24 hr, weekly, annual) average levels that might be associated with increased respiratory illness in young children cannot be clearly derived based alone on the limited monitoring data reported by Spengler et al. (1979). Rather, additional data from other studies of NO_2 levels typically encountered in American homes using gas stoves may also be of value in attempting to estimate the effective NO_2 exposure parameters. The results of several such studies are summarized in Table 15-7, along with the Spengler et al. (1979) findings. Collectively, the studies found daily peak 1-2 hr NO_2 levels generally to average between 0.04 and 0.50 ppm (depending on room of house monitored) but some instantaneous peak levels in kitchens were at times as high as 1.0 ppm); whereas 24-hr and annual average levels fell in the range of 0.02 to 0.07 ppm, and 0.02 to 0.06 ppm, respectively. Reported increases in respiratory disease symptoms in young children residing in gas stove homes could be associated with any of these NO_2 exposure levels/durations.

Placing such levels in perspective against reported ambient air levels in the United States, examination of selected nationwide monitoring data for 1975 to 1980 (discussed in Chapter 8) reveals that peak 1-hr NO_2 concentrations only occassionally equalled or exceeded 0.4 ppm in a few locations nationwide (e.g., Los Angeles; several other California sites; Ashland, Kentucky; and Port Huron, Michigan). Similarly, annual average NO_2 levels during the 1975 to 1980 period exceeded 0.05 ppm in some areas, including such heavily populated locations as Anaheim and San Diego, California, and Chicago, Illinois.

15.6.1.3 Chronic Respiratory Disease Effects--A number of investigators have failed to find an association between long-term mean ambient NO_2 exposure and the prevalence of chronic respiratory disease in adults. In contrast, long term NO_2 concentrations have been reported to be associated with mortality, various cancers, and arteriosclerotic heart disease. Such epidemiologic results, however, are not consistent in their findings; one even showed a significant negative correlation between NO_2 and mortality. In addition, the quality of data available for such long-term studies is such that the results must be viewed with much skepticism until they can be substantiated by additional research.

TABLE 15-7. NITROGEN DIOXIDE LEVELS REPORTED IN GAS AND ELECTRIC STOVE HOMES

Site and conditions	Parameter	NO_2 concentration (ppm)			Measurement method	Reference
		Home 1: 2000 ft. split-level with well ventilated kitchen.	Home 3: 2-story apartment with small, unventilated kitchen.	Home 4: 1500 ft^2 ranch style with kitchen open to other rooms.		
		range (mean)	range (mean)	range (mean)		
Kitchen, 1 meter from stove	Daily peak 2-hr avg	.04-.28 (.10)	.03-.31 (.10)	.05-.41 (.18)	Chemiluminesent Analyzer	Cote, Wade, and Yocom, 1974 Suburban homes in Connecticut
	24-hr avg	.01-.08 (.04)	.03-.13 (.06)	.04-.11 (.06)		
Living room	Daily peak 2-hr avg	.01-.17 (.06)	.01-.06 (.04)	.04-.35 (.11)		
	24-hr avg	.01-.05 (.03)	.01-.04 (.03)	.03-.07 (.04)		
Bedroom	Daily peak 2-hr avg	.02-.09 (.05)	.02-.19 (.07)			
	24-hr avg	.02-.04 (.03)	.03-.05 (.03)			
Outdoors	Daily peak 2-hr avg	.02-.08 (.04)	.01-.09 (.04)	.02-.12 (.05)		
	24-hr avg	.01-.05 (.02)	.02-.03 (.02)	.01-.04 (.02)		
Study simulated typical gas stove use patterns					Chemiluminesent Analyzer	Hollowel et al., 1980 Energy efficient research house, .33-.44 air changes per hour (ach).
Kitchen	Peak 1-hr avg		.45			
	24-hr avg		.07			
Living room	Peak 1-hr avg		.40			
	24-hr avg		.07			
Bedroom	Peak 1-hr avg		.24			
	24-hr avg		.05			
Outdoors	Peak 1-hr avg		.07			
	24-hr avg		.04			

TABLE 15-7. (continued)

Site and conditions	Parameter	NO_2 concentration (ppm)	Measurement method	Reference
Kitchen with a gas oven on for 1-hr at 350°F			Chemiluminescent Analyzer	Hollowel et al., 1978 Test kitchen ($27m^3$)
0.25 ach (no stove vent)	1-hr average	1.20		
1.0 ach (hood vent above stove)	1-hr average	0.80		
2.5 ach (hood vent with fan at 50 CFM)	1-hr average	0.40		
7.0 ach (hood vent with fan at 140 CFM)	1-hr average	0.10		
Outside during test	1-hr average	0.03		
		range (mean)		
83 gas stove homes	24-hr average	.01-.11 (0.05)	Modified Jacobs-Hochheiser (arsenite modified)	Keller et al., 1979 Gas and electric stove homes in Columbus, Ohio
50 electric stove homes	24-hr average	0-.06 (.02)		
53 outdoor samples in vicinity	24-hr average	.02-.05 (.03)		
46 gas stove	continuous measurement over 3 day periods	peak values in some homes exceeded 1 ppm; peaks during cooking reached as high as 8 x 24-hr average	Chemiluminescent Analyzer	
Activity room (gas stove homes)	95th percentile of 24-hr averages measured over a 1 year period	.02-.06	Modified Sodium Arsenite	Speizer et al., 1980 Activity room in 5-11 gas and electric stove homes in each of six communities. Also monitored in 1 kitchen of a gas stove home for 2 wks.
Activity room (electric stove homes)		.01-.05		
Outdoors		.01-.06		
Kitchen, 3-ft from gas stove home	continuous	peak concentrations in the range of 0.25-0.50 were observed for 10-15 minute periods during oven or stove use.	Chemiluminescent Analyzer	

15.6.1.4 Sensory System Effects--In addition to pulmonary function and respiratory disease effects, NO_2 exposures have been shown to exert effects on sensory systems. This includes detection of NO_2 as a pungent odor at concentrations as low as 210 $\mu g/m^3$ (0.11 ppm) of NO_2 immediately upon exposure. Under higher exposure conditions (10 ppm) impaired odor detection occurs. Both of these sensory effects, however, appear to be of negligible health concern in view of their temporary, reversible nature and generally minor impact on normal human functions and activities. Probably of somewhat greater importance are certain other NO_2 sensory effects, especially impairment of dark adaption which can occur in human subjects at NO_2 levels as low as 130 to 150 $\mu g/m^3$ (.07 to .08 ppm). It is difficult to appraise fully the health significance of such an effect, however; but it appears to be of generally negligible health concern except, perhaps, for certain occupational or public safety situations where rapid dark adaptation may be important.

15.6.2 Effects of NO_x-Derived Compounds

Nitrate poisoning (i.e., the formation of sufficient methaemoglobin to produce cyanosis) occurs not infrequently in the United States, most often in children. It is not believed, however, that the inhalation of atmospheric nitrates is important in producing symptoms since the quantity absorbed from the air would represent a relatively small fraction of that ingested by other routes (e.g., food and water). Even if absorption from the lung was several times that from the gut, atmospheric nitrates probably would still be unimportant in this regard.

Nitrate aerosols and nitric acid vapor may represent significant respiratory irritants. Two studies have suggested that increases in atmospheric nitrate concentrations or combined nitrate and sulfate concentrations were associated with increases in asthma attacks. A laboratory study, however, indicated that short-term exposure to sodium nitrate at concentrations at least 100 times the total nitrate in ambient exposures had no effect on pulmonary function in healthy individuals or in asthmatics.

Chemical reactions in the atmosphere involving NO_2 and hydrocarbons can produce peroxyacetyl nitrate (PAN), a strong eye irritant. The effects of PAN have been thoroughly reviewed recently in the document Air Quality Criteria for Ozone and Other Photochemical Oxidants (U.S. EPA, 1978) and reference is therefore made to this document for further information.

Although the existence of a mechanism for the nitrosation *in vivo* of an exogenous amine, morpholine, by inhaled NO_2 has recently been demonstrated in mice, there is no evidence, to date, that nitrogenous atmospheric pollutants contribute to the *in vivo* formation of nitrosamines in humans or that nitrosamines inhaled from the ambient air represent significant health hazards.

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APPENDIX A: GLOSSARY

$AaDO_2$: Alveolar-arterial difference or gradient of the partial pressure ot oxygen. An overall measure of the efficiency of the lung as a gas exchanger. In healthy subjects, the gradient is 5 to 15 mm Hg (torr).

A/PR/8 virus: A type of virus capable of causing influenza in laboratory animals; also, A/PR/8/34.

Abscission: The process whereby leaves, leaflets, fruits, or other plant parts become detached from the plant.

Absorption coefficient: A quantity which characterizes the attenuation with distance of a beam of electromagnetic radiation (like light) in a substance.

Absorption spectrum: The spectrum that results after any radiation has passed through an absorbing substance.

Abstraction: Removal of some constituent of a substance or molecule.

Acetaldehyde: CH_3CHO; an intermediate in yeast fermentation of carbohydrate and in alcohol metabolism; also called acetic aldehyde, ethaldehyde, ethanal.

Acetate rayon: A staple or filament fiber made by extrusion of cellulose acetate. It is saponified by dilute alkali whereas viscose rayon remains unchanged.

Acetylcholine: A naturally-occurring substance in the body which can cause constriction of the bronchi in the lungs.

Acid: A substance that can donate hydrogen ions.

Acid dyes: A large group of synthetic coal tar-derived dyes which produce bright shades in a wide color range. Low cost and ease of application are features which make them the most widely used dyes for wool. Also used on nylon. The term acid dye is derived from their precipitation in an acid bath.

Acid mucopolysaccharide: A class of compounds composed of protein and polysaccharide. Mucopolysaccharides comprise much of the substance of connective tissue.

Acid phosphatase: An enzyme (EC 3.1.3.2) which catalyzes the disassociation of phosphate (PO_4) from a wide range of monoesters of orthophosphoric acid. Acid phosphatase is active in an acidic pH range.

Acid rain: Rain having a pH less than 5.6, the minimum expected from atmospheric CO_2.

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Acrolein: $CH_2=CHCHO$; a volatile, flammable, oily liquid, giving off irritant vapor. Strong irritant of skin and mucuous membranes. Also called acrylic aldehyde, 2-propenal.

Acrylics (plastics): Plastics which are made from acrylic acid and are light in weight, have great breakage resistance, and a lack of odor and taste. Not resistant to scratching, burns, hot water, alcohol or cleaning fluids. Examples include Lucite and Plexiglass. Acrylics are thermoplastics and are softened by heat and hardened into definite shapes by cooling.

Acrylic fiber: The generic name of man-made fibers derived from acrylic resins (minimum of 85 percent acrylonitrite units).

Actinic: A term applied to wavelengths of light too small to affect one's sense of sight, such as ultraviolet.

Actinomycetes: Members of the genus Actinomyces; nonmotile, nonspore-forming, anaerobic bacteria, including both soil-dwelling saprophytes and disease-producing parasites.

Activation energy: The energy required to bring about a chemical reaction.

Acute respiratory disease: Respiratory infection, usually with rapid onset and of short duration.

Acute toxicity: Any poisonous effect produced by a single short-term exposure, that results in severe biological harm or death.

Acyl: Any organic radical or group that remains intact when an organic acid forms an ester.

Adenoma: An ordinarily benign neoplasm (tumor) of epithelial tissue; usually well circumscribed, tending to compress adjacent tissue rather than infiltrating or invading.

Adenosine monophosphate (AMP): A nucleotide found amoung the hydrolysis products of all nucleic acids; also called adenylic acid.

Adenosine triphosphatase (ATPase): An enzyme (EC 3.6.1.3) in muscle and elsewhere that catalyzes the release of the high-energy, terminal phosphate group of adenosine triphosphate.

Adrenalectomy: Removal of an adrenal gland. This gland is located near or upon the kidney and is the site of origin of a number of hormones.

Adsorption: Adhesion of a thin layer of molecules to a liquid or solid surface.

Advection: Horizontal flow of air at the surface or aloft; one of the means by which heat is transferred from one region of the earth to another.

Aerodynamic diameter: Expression of aerodynamic behavior of an irregularly shaped particle in terms of the diameter of a sphere of unit density having identical aerodynamic behavior to the particle in question.

Aerosol: Solid particles or liquid droplets which are dispersed or suspended in a gas.

Agglutination: The process by which suspended bacteria, cells or similar particles adhere and form into clumps.

Airborne pathogen: A disease-causing microorganism which travels in the air or on particles in the air.

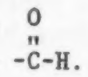
Air pollutant: A substance present in the ambient atmosphere, resulting from the activity of man or from natural processes, which may cause damage to human health or welfare, the natural environment, or materials or objects.

Airway conductance: Inverse of airway resistance.

Airway resistance (R_{aw}): The pressure difference between the alveoli and the mouth required to produce an air flow of 1 liter per second.

Alanine aminotransferase: An enzyme (EC 2.6.1.2) transferring amino groups from L-alanine to 2-ketoglutarate. Also known as alanine transaminase.

Albumin: A type of simple, water-soluble protein widely distributed throughout animal tissues and fluids, particularly serum.

Aldehyde: An organic compound characterized by the group  $-\overset{O}{\overset{\|}{C}}-H$.

Aldolase: An enzyme (EC 4.1.2.7) involved in metabolism of fructose which catalyzes the formation of two 3-carbon intermediates in the major pathway of carbohydrate metabolism.

Algal bloom: Sudden spurt in growth of algae which can affect water quality adversely.

Alkali: A salt of sodium or potassium capable of neutralizing acids.

Alkaline phosphatase: A phosphatase (EC 3.1.3.1) with an optimum pH of 8.6, present ubiquitously.

Allergen: A material that, as a result of coming into contact with appropriate tissues of an animal body, induces a state of sensitivity resulting in various reactions; generally associated with idiosyncratic hypersensitivities.

Alpha-hydroxybutyrate dehydrogenase: An enzyme (EC 1.1.1.30), present mainly in mitochondria, which catalyzes the conversion of hydroxybutyrate to acetoacetate in intermediate biochemical pathways.

Alpha rhythm: A rhythmic pulsation obtained in brain waves exhibited in the sleeping state of an individual.

Alveolar capillary membrane: Finest portion of alveolar capillaries, where gas transfer to and from blood takes place.

Alveolar macrophages (AM): Large, mononuclear, phagocytic cells found on the alveolar surface, responsible for the sterility of the lung.

Alveolar oxygen partial pressure (PAO_2): Partial pressure of oxygen in the air contained in the air sacs of the lungs.

Alveolar septa: The tissue between two adjacent pulmonary alveoli, consisting of a close-meshed capillary network covered on both surfaces by thin alveolar epithelial cells.

Alveolus: An air cell; a terminal, sac-like dilation in the lung. Gas exchange (O_2/CO_2) occurs here.

Ambient: The atmosphere to which the general population may be exposed. Construed here not to include atmospheric conditions indoors, or in the workplace.

Amine: A substance that may be derived from ammonia (NH_3) by the replacement of one, two or three of the hydrogen (H) atoms by hydrocarbons or other radicals (primary, secondary or tertiary amines, respectively).

Amino acids: Molecules consisting of a carboxyl group, a basic amino group, and a residue group attached to a central carbon atom. Serve as the building blocks of proteins.

p-Aminohippuric acid (PAH): A compound used to determine renal plasma flow.

Aminotriazole: A systemic herbicide, $C_2H_4N_4$, used in areas other than croplands, that also possesses some antithyroid activity; also called amitrole.

Ammonification: Decomposition with production of ammonia or ammonium compounds, esp. by the action of bacteria on nitrogenous organic matter.

Ammonium: Anion (NH_4^+) or radical (NH_4) derived from ammonia by combination with hydrogen. Present in rainwater, soils and many commercial fertilizers.

Amnestic: Pertains to immunologic memory: upon receiving a second dose of antigen, the host "remembers" the first dose and responds faster to the challenge.

Anaerobic: Living, active or occurring in the absence of free oxygen.

Anaerobic bacteria: A type of microscopic organism which can live in an environment not containing free oxygen.

Anaphylactic dyspneic attack: Difficulty in breathing associated with a systemic allergic response.

Anaphylaxis: A term commonly used to denote the immediate, transient kind of immunological (allergic) reaction characterized by contraction of smooth muscle and dilation of capillaries due to release of pharmacologically active substances.

Angiosperm: A plant having seeds enclosed in an ovary; a flowering plant.

Angina pectoris: Severe constricting pain in the chest which may be caused by depletion of oxygen delivery to the heart muscle; usually caused by coronary disease.

Angstrom (Å): A unit (10^{-8} cm) used in the measurement of the wavelength of light.

Anhydride: A compound resulting from removal of water from two molecules of a carboxylic (-COOH) acid. Also, may refer to those substances (anhydrous) which do not contain water in chemical combination.

Anion: A negatively charged atom or radical.

Anorexia: Diminished appetite; aversion to food.

Anoxic: Without or deprived of oxygen.

Anthraquinone: A yellow crystalline ketone, $C_{14}H_8O_2$, derived from anthracene and used in the manufacture of dyes.

Anthropogenic: Of, relating to or influenced by man. An anthropogenic source of pollution is one caused by man's actions.

Antibody: Any body or substance evoked by the stimulus of an antigen and which reacts specifically with antigen in some demonstrable way.

Antigen: A material such as a foreign protein that, as a result of coming in contact with appropriate tissues of an animal, after a latent period, induces a state of sensitivity and/or the production of antibody.

Antistatic agent: A chemical compound applied to fabrics to reduce or eliminate accumulation of static electricity.

Arachidonic acid: Long-chain fatty-acid which serves as a precursor of prostaglandins.

Area source: In air pollution, any small individual fuel combustion or other pollutant source; also, all such sources grouped over a specific area.

Aromatic: Belonging to that series of carbon-hydrogen compounds in which the carbon atoms form closed rings containing unsaturated bonds (as in benzene).

Arterial partial pressure of oxygen (PaO_2): Portion of total pressure of dissolved gases in arterial blood as measured directly from arterial blood.

Arterialized partial pressure of oxygen: The portion of total pressure of dissolved gases in arterial blood attributed to oxygen, as measured from non-arterial (e.g., ear-prick) blood.

Arteriosclerosis: Commonly called hardening of the arteries. A condition that exists when the walls of the blood vessels thicken and become infiltrated with excessive amounts of minerals and fatty materials.

Artifact: A spurious measurement produced by the sampling or analysis process.

Ascorbic acid: Vitamin C, a strong reducing agent with antioxidant properties.

Aspartate transaminase: Also known as aspartate aminotransferase (EC 2.6.1.1). An enzyme catalyzing the transfer of an amine group from glutamic acid to oxaloacetic, forming aspartic acid in the process. Serum level of the enzyme is increased in myocardial infarction and in diseases involving destruction of liver cells.

Asphyxia: Impaired exchange of oxygen and carbon dioxide, excess of carbon dioxide and/or lack of oxygen, usually caused by ventilatory problems.

Asthma: A term currently used in the context of bronchial asthma in which there is widespread narrowing of the airways of the lung. It may be aggravated by inhalation of pollutants and lead to "wheezing" and shortness of breath.

Asymptomatic: Presenting no subjective evidence of disease.

Atmosphere: The body of air surrounding the earth. Also, a measure of pressure (atm.) equal to the pressure of air at sea level, 14.7 pounds per square inch.

Atmospheric deposition: Removal of pollutants from the atmosphere onto land, vegetation, water bodies or other objects, by absorption, sedimentation, Brownian diffusion, impaction, or precipitation in rain.

Atomic absorption spectrometry: A measurement method based on the absorption of radiant energy by gaseous ground-state atoms. The amount of absorption depends on the population of the ground state which is related to the concentration of the sample being analyzed.

Atropine: A poisonous white crystalline alkaloid, $C_{17}H_{23}NO_3$, from belladonna and related plants, used to relieve spasms and to dilate the pupil of the eye.

Autocorrelation: Statistical interdependence of variables being analyzed; produces problems, for example, when observations may be related to previous measurements or other conditions.

Autoimmune disease: A condition in which antibodies are produced against the subject's own tissues.

Autologous: A term referring to cellular elements, such as red blood cells and alveolar macrophage, from the same organism; also, something natually and normally ocurring in some part of the body.

Autotrophic: A term applied to those microorganisms which are able to maintain life without an exogenous organic supply of energy, or which only need carbon dioxide or carbonates and simple inorganic nitrogen.

Autotrophic bacteria: A class of microorganisms which require only carbon dioxide or carbonates and a simple inorganic nitrogen compound for carrying on life processes.

Auxin: An organic substance that causes lengthening of the stem when applied in low concentrations to shoots of growing plants.

Awn: One of the slender bristles that terminate the glumes of the spikelet in some cereals and other grasses.

Azo dye: Dyes in which the azo group is the chromophore and joins benzene or napthalene rings.

Background measurement: A measurement of pollutants in ambient air due to natural sources; usually taken in remote areas.

Bactericidal activity: The process of killing bacteria.

Barre: Bars or stripes in a fabric, caused by uneven weaving, irregular yarn or uneven dye distribution.

Basal cell: One of the innermost cells of the deeper epidermis of the skin.

Benzenethiol: A compound of benzene and a hydrosulfide group.

Beta (b)-lipoprotein: A biochemical complex or compound containing both lipid and protein and characterized by having a large molecular weight, rich in cholesterol. Found in certain fractions of human plasma.

Bilateral renal sclerosis: A hardening of both kidneys of chronic inflammatory origin.

Biomass: That part of a given habitat consisting of living matter.

Biosphere: The part of the earth's crust, waters and atmosphere where living organisms can subsist.

Biphasic: Having two distinct successive stages.

Bleb: A collection of fluid beneath the skin; usually smaller than bullae or blisters.

Blood urea: The chief end product of nitrogen metabolism in mammals, excreted in human urine in the amount of about 32 grams (1 oz.) a day.

Bloom: A greenish-gray appearance imparted to silk and pile fabrics either by nature of the weave or by the finish; also, the creamy white color observed on some good cottons.

Blue-green algae: A group of simple plants which are the only N_2-fixing organisms which photosynthesize as do higher plants.

Brightener: A compound such as a dye, which adheres to fabrics in order to provide better brightness or whiteness by converting ultraviolet radiation to visible light. Sometimes called optical bleach or whitening agent. The dyes used are of the florescent type.

Broad bean: The large flat edible seed of an Old World upright vetch (Vicia faba), or the plant itself, widely grown for its seeds and for fodder.

Bronchi: The first subdivisions of the trachea which conduct air to and from the bronchioles of the lungs.

Bronchiole: One of the finer subdivisions of the bronchial (trachea) tubes, less than 1 mm in diameter, and having no cartilage in its wall.

Bronchiolitis: Inflammation of the smallest bronchial tubes.

Bronchiolitis fibrosa obliterans syndrome: Obstruction of the bronchioles by fibrous granulation arising from an ulcerated mucosa; the condition may follow inhalation of irritant gases.

Bronchitis: Inflammation of the mucous membrane of the bronchial tubes. It may aggravate an existing asthmatic condition.

Bronchoconstrictor: An agent that causes a reduction in the caliber (diameter) of a bronchial tube.

Bronchodilator: An agent which causes an increase in the caliber (diameter) of a bronchus or bronchial tube.

Bronchopneumonia: Acute inflammation of the walls of the smaller bronchial tubes, with irregular area of consolidation due to spread of the inflammation into peribronchiolar alveoli and the alveolar ducts.

Brownian diffusion: Diffusion by random movement of particles suspended in liquid or gas, resulting from the impact of molecules of the fluid surrounding the particles.

Buffer: A substance in solution capable of neutralizing both acids and bases and thereby maintaining the original pH of the solution.

Buffering capacity: Ability of a body of water and its watershed to neutralize introduced acid.

Butanol: A four-carbon, straight-chain alcohol, C_4H_9OH, also known as butyl alcohol.

Butylated hydroxytoluene (BHT): A crystalline phenolic antioxidant.

Butylated hydroxyanisol (BHA): An antioxidant.

^{14}C labeling: Use of a radioactive form of carbon as a tracer, often in metabolic studies.

^{14}C-proline: An amino acid which has been labeled with radioactive carbon.

Calcareous: Resembling or consisting of calcium carbonate (lime), or growing on limestone or lime-containing soils.

Calorie: Amount of heat required to raise temperature of 1 gram of water at 15°C by 1 degree.

Cannula: A tube that is inserted into a body cavity, or other tube or vessel, usually to remove fluid.

Capillary: The smallest type of vessel; resembles a hair. Usually in reference to a blood or lymphatic capillary vessel.

Carbachol: A chemical compound (carbamoylcholine chloride, $C_6H_{15}ClN_2O_2$) that produces a constriction of the bronchi; a parasympathetic stimulant used in veterinary medicine and topically in glaucoma.

Carbon monoxide: An odorless, colorless, toxic gas with a strong affinity for hemoglobin and cytochrome; it reduces oxygen absorption capacity, transport and utilization.

Carboxyhemoglobin: A fairly stable union of carbon monoxide with hemoglobin which interferes with the normal transfer of carbon dioxide and oxygen during circulation of blood. Increasing levels of carboxyhemoglobin result in various degrees of asphyxiation, including death.

Carcinogen: Any agent producing or playing a stimulatory role in the formation of a malignancy.

Carcinoma: Malignant new growth made up of epithelial cells tending to infiltrate the surrounding tissues and giving rise to metastases.

Cardiac output: The volume of blood passing through the heart per unit time.

Cardiovascular: Relating to the heart and the blood vessels or the circulation.

Carotene: Lipid-soluble yellow-to-orange-red pigments universally present the photosynthetic tissues of higher plants, algae, and the photosynthetic bacteria.

Cascade impactor: A device for measuring the size distribution of particulates and/or aerosols, consisting of a series of plates with orifices of graduated size which separate the sample into a number of fractions of decreasing aerodynamic diameter.

Catabolism: Destructive metabolism involving the release of energy and resulting in breakdown of complex materials in the organism.

Catalase: An enzyme (EC 1.11.1.6) catalyzing the decomposition of hydrogen peroxide to water and oxygen.

Catalysis: A modification of the rate of a chemical reaction by some material which is unchanged at the end of the reaction.

Catalytic converter: An air pollution abatement device that removes organic contaminants by oxidizing them into carbon dioxide and water.

Catecholamine: A pyrocatechol with an alkalamine side chain, functioning as a hormone or neurotransmitter, such as epinephrine, morepinephrine, or dopamine.

Cathepsins: Enzymes which have the ability to hydrolyze certain proteins and peptides; occur in cellular structures known as lysosomes.

Cation: A positively charged ion.

Cellular permeability: Ability of gases to enter and leave cells; a sensitive indicator of injury to deep-lung cells.

Cellulose: The basic substance which is contained in all vegetable fibers and in certain man-made fibers. It is a carbohydrate and constitutes the major substance in plant life. Used to make cellulose acetate and rayon.

Cellulose acetate: Commonly refers to fibers or fabrics in which the cellulose is only partially acetylated with acetate groups. An ester made by reacting cellulose with acetic anhydride with SO_4 as a catalyst.

Cellulose rayon: A regenerated cellulose which is chemically the same as cellulose except for physical differences in molecular weight and crystallinity.

Cellulose triacetate: A cellulose fiber which is completely acetylated. Fabrics of triacetate have higher heat resistance than acetate and may be safely ironed at higher temperature. Such fabrics have improved ease-of-care characteristics because after heat treatment during manufacture, a change in the crystalline structure of the fiber occurs.

Cellulosics: Cotton, viscose rayon and other fibers made of natural fiber raw materials.

Celsius scale: The thermometric scale in which freezing point of water is 0 and boiling point is 100.

Central hepatic necrosis: The pathologic death of one or more cells, or of a portion of the liver, involving the cells adjacent to the central veins.

Central nervous system (CNS): The brain and the spinal cord.

Centroacinar area: The center portion of a grape-shaped gland.

Cerebellum: The large posterior brain mass lying above the pons and medulla and beneath the posterior portion of the cerebrum.

Cerebral cortex: The layer of gray matter covering the entire surface of the cerebral hemisphere of mammals.

Chain reaction: A reaction that stimulates its own repetition.

Challenge: Exposure of a test organism to a virus, bacteria, or other stress-causing agent, used in conjunction with exposure to a pollutant of interest, to explore possible susceptibility brought on by the pollutant.

Chamber study: Research conducted using a closed vessel in which pollutants are reacted or substances exposed to pollutants.

Chemiluminescence: A measurement technique in which radiation is produced as a result of chemical reaction.

Chemotactic: Relating to attraction or repulsion of living protoplasm by chemical stimuli.

Chlorophyll: A group of closely related green photosynthetic pigments occurring in leaves, bacteria, and organisms.

Chloroplast: A plant cell inclusion body containing chlorophyll.

Chlorosis: Discoloration of normally green plant parts that can be caused by disease, lack of nutrients, or various air pollutants, resulting in the failure of chlorophyll to develop.

Cholesterol: A steroid alcohol $C_{27}H_{45}OH$; the most abundant steroid in animal cells and body fluids.

Cholinesterase (CHE): One (EC 3.1.1.8) of a family of enzymes capable of catalyzing the hydrolysis of acylcholines.

Chondrosarcoma: A malignant neoplasm derived from cartilage cells, occurring most frequently near the ends of long bones.

Chromatid: Each of the two strands formed by longitudinal duplication of a chromosome that becomes visible during an early stage of cell division.

Chromophore: A chemical group that produces color in a molecule by absorbing near ultraviolet or visible radiation when bonded to a nonabsorbing, saturated residue which possesses no unshared, nonbonding valence electrons.

Chromosome: One of the bodies (46 in man) in the cell nucleus that is the bearer and carrier of genetic information.

Chronic respiratory disease (CRD): A persistent or long-lasting intermittent disease of the respiratory tract.

Cilia: Motile, often hairlike extensions of a cell surface.

Ciliary action: Movements of cilia in the upper respiratory tract, which move mucus and foreign material upward.

Ciliogenesis: The formation of cilia.

Citric acid (Krebs) cycle: A major biochemical pathway in cells, involving terminal oxidation of fatty acids and carbohydrates. It yields a major portion of energy needed for essential body functions and is the major source of CO_2. It couples the glycolytic breakdown of sugar in the cytoplasm with those reactions producing ATP in the mitochondria. It also serves to regulate the synthesis of a number of compounds required by a cell.

Clara cell: A nonciliated mammalian cell.

Closing volume (CV): The lung volume at which the flow from the lower parts of the lungs becomes severely reduced or stops during expiration, presumably because of airway closure.

Codon: A sequence of three nucleotides which encodes information required to direct the synthesis of one or more amino acids.

Coefficient of haze (COH): A measurement of visibility interference in the atmosphere.

Cohort: A group of subjects included in a test or experiment; usually characterized by age, class or other characteristic.

Collagen: The major protein of the white fibers of connective tissue, cartilage, and bond. Comprises over half the protein of the mammal.

Collisional deactivation: Reduction in energy of excited molecules caused by collision with other molecules or other objects such as the walls of a container.

Colorimetric: A chemical analysis method relying on measurement of the degree of color produced in a solution by reaction with the pollutant of interest.

Community exposure: A situation in which people in a sizeable area are subjected to ambient pollutant concentrations.

Compliance: A measure of the change in volume of an internal organ (e.g. lung, bladder) produced by a unit of pressure.

Complement: Thermolabile substance present in serum that is destructive to certain bacteria and other cells which have been sensitized by specific complement-fixing antibody.

Compound: A substance with its own distinct properties, formed by the chemical combination of two or more elements in fixed proportion.

Concanavalin-A: One of two crystalline globulins occurring in the jack bean; a potent hemagglutinin.

Conifer: A plant, generally evergreen, needle-leafed, bearing naked seeds singly or in cones.

Converter: See catalytic converter.

Coordination number: The number of bonds formed by the central atom in a complex.

Copolymer: The product of the process of polymerization in which two or more monomeric substances are mixed prior to polymerization. Nylon is a copolymer.

Coproporphyrin: One of two porphyrin compounds found normally in feces as a decomposition product of bilirubin (a bile pigment). Porphyrin is a widely-distributed pigment consisting of four pyrrole nuclei joined in a ring.

Cordage: A general term which includes banding, cable, cord, rope, string, and twine made from fibers. Synthetic fibers used in making cordage include nylon and dacron.

Corrosion: Destruction or deterioration of a material because of reaction with its environment.

Corticosterone: A steroid obtained from the adrenal cortex. It induces some deposition of glycogen in the liver, sodium conservation, and potassium excretion.

Cosmopolitan: In the biological sciences, a term denoting worldwide distribution.

Coulometric: Chemical analysis performed by determining the amount of a substance released in electrolysis by measuring the number of coulombs used.

Coumarin: A toxic white crystalline lactone ($C_9H_6O_2$) found in plants.

Coupler: A chemical used to combine two others in a reaction, e.g. to produce the azo dye in the Griess-Saltzman method for NO_2.

Crevice corrosion: Localized corrosion occurring within crevices on metal surfaces exposed to corrosives.

Crosslink: To connect, by an atom or molecule, parallel chains in a complex chemical molecule, such as a polymer.

Cryogenic trap: A pollutant sampling method in which a gaseous pollutant is condensed out of sampled air by cooling (e.g. traps in one method for nitrosamines are maintained below $-79^\circ C$, using solvents maintained at their freezing points).

Cuboidal: Resembling a cube in shape.

Cultivar: An organism produced by parents belonging to different species or to different strains of the same species, originating and persisting under cultivation.

Cuticle: A thin outer layer, such as the thin continuous fatty film on the surface of many higher plants.

Cyanosis: A dark bluish or purplish coloration of the skin and mucous membrane due to deficient oxygenation of the blood.

Cyclic GMP: Guanosine 5'-phosphoric acid.

Cytochrome: A class of hemoprotein whose principal biological function is electron and/or hydrogen transport.

Cytology: The anatomy, physiology, pathology and chemistry of the cell.

Cytoplasm: The substance of a cell exclusive of the nucleus.

Dacron: The trade name for polyester fibers made by E.I. du Pont de Nemours and Co., Inc., made from dimethyl terephthalate and ethylene glycol.

Dark adaptation: The process by which the eye adjusts under reduced illumination and the sensitivity of the eye to light is greatly increased.

Dark respiration: Metabolic activity of plants at night; consuming oxygen to use stored sugars and releasing carbon dioxide.

Deciduous plants: Plants which drop their leaves at the end of the growing season.

Degradation (textiles): The decomposition of fabric or its components or characteristics (color, strength, elasticity) by means of light, heat, or air pollution.

Denitrification: A bacterial process occurring in soils, or water, in which nitrate is used as the terminal electron acceptor and is reduced primarily to N_2. It is essentially an anaerobic process; it can occur in the presence of low levels of oxygen only if the microorganisms are metabolizing in an anoxic microzone.

De novo: Over again.

Deoxyribonucleic acid (DNA): A nucleic acid considered to be the carrier of genetic information coded in the sequence of purine and pyrimidine bases (organic bases). It has the form of a double-stranded helix of a linear polymer.

Depauperate: Falling short of natural development or size.

Derivative spectrophotometer: An instrument with an increased capability for detecting overlapping spectral lines and bands and also for suppressing instrumentally scattered light.

Desorb: To release a substance which has been taken into another substance or held on its surface; the opposite of absorption or adsorption.

Desquamation: The shedding of the outer layer of any surface.

Detection limit: A level below which an element or chemical compound cannot be reliably detected by the method or measurement being used for analysis.

Detritus: Loose material that results directly from disintegration.

DeVarda alloy: An alloy of 50 percent Cu, 45 percent Al, 5 percent Zn.

Diastolic blood pressure: The blood pressure as measured during the period of filling the cavities of the heart with blood.

Diazonium salt: A chemical compound (usually colored) of the general structure $ArN_2^+Cl^-$, where Ar refers to an aromatic group.

Diazotizer: A chemical which, when reacted with amines (RNH_2, for example), produces a diazonium salt (usually a colored compound).

Dichotomous sampler: An air-sampling device which separates particulates into two fractions by particle size.

Differentiation: The process by which a cell, such as a fertilized egg, divides into specialized cells, such as the embryonic types that eventually develop into an entire organism.

Diffusion: The process by which molecules or other particles intermingle as a result of their random thermal motion.

Diffusing capacity: Rate at which gases move to or from the blood.

Dimer: A compound formed by the union of two like radicals or molecules.

Dimerize: Formation of dimers.

1,6-diphosphofructose aldolase: An enzyme (EC 4.1.1.13) cleaving fructose 1,6-bisphosphate to dihydroxyacetone phosphate and glyceraldehyde-3-phosphate.

D-2,3-diphosphoglycerate: A salt or ester of 2,3-diphosphoglyceric acid, a major component of certain mammalian erythrocytes involved in the release of O_2 from HbO_2. Also a postulated intermediate in the biochemical pathway involving the conversion of 3- to 2-phosphoglyceric acid.

Diplococcus pneumoniae: A species of spherical-shaped bacteria belonging to the genus Streptococcus. May be a causal agent in pneumonia.

Direct dye: A dye with an affinity for most fibers; used mainly when color resistance to washing is not important.

Disperse dyes: Also known as acetate dyes; these dyes were developed for use on acetate fabrics, and are now also used on synthetic fibers.

Distal: Far from some reference point such as median line of the body, point of attachment or origin.

Diurnal: Having a repeating pattern or cycle 24 hours long.

DL_{CO}: The diffusing capacity of the lungs for carbon monoxide. The ability of the lungs to transfer carbon monoxide from the alveolar air into the pulmonary capillary blood.

Dorsal hyphosis: Abnormal curvative of the spine; hunch-back.

Dose: The quantity of a substance to be taken all at one time or in fractional amounts within a given period; also the total amount of a pollutant delivered or concentration per unit time times time.

Dose-response curve: A curve on a graph based on responses occurring in a system as a result of a series of stimuli intensities or doses.

Dry deposition: The processes by which matter is transferred to ground from the atmosphere, other than precipitation; includes surface absorption of gases and sedimentation, Brownian diffusion and impaction of particles.

Dyeing: A process of coloring fibers, yarns, or fabrics with either natural or synthetic dyes.

Dynamic calibration: Testing of a monitoring system using a continuous sample stream of known concentration.

Dynamic compliance (C_{Ldyn}): Volume change per unit of transpulmonary pressure minus the pressure of pulmonary resistance during airflow.

Dynel: A trademark for a modacrylic staple fiber spun from a copolymer of acrylonitrile and vinyl chloride. It has high strength, quick-drying properties, and resistance to alkalies and acids.

Dyspepsia: Indigestion, upset stomach.

Dyspnea: Shortness of breath; difficulty or distress in breathing; rapid breathing.

Ecosystem: The interacting system of a biological community and its environment.

Eddy: A current of water or air running contrary to the main current.

Edema: Pressure of excess fluid in cells, intercellular tissue or cavities of the body.

Elastomer: A synthetic rubber product which has the physical properties of natural rubber.

Electrocardiogram: The graphic record of the electrical currents that initiate the heart's contraction.

Electrode: One of the two extremities of an electric circuit.

Electrolyte: A non-metallic electric conductor in which current is carried by the movement of ions; also a substance which displays these qualities when dissolved in water or another solvent.

Electronegativity: Measure of affinity for negative charges or electrons.

Electron microscopy: A technique which utilizes a focused beam of electrons to produce a high-resolution image of minute objects such as particulate matter, bacteria, viruses, and DNA.

Electronic excitation energy: Energy associated in the transition of electrons from their normal low-energy orbitals or orbitals of higher energy.

Electrophilic: Having an affinity for electrons.

Electrophoresis: A technique by which compounds can be separated from a complex mixture by their attraction to the positive or negative pole of an applied electric potential.

Eluant: A liquid used in the process of elution.

Elute: To perform an elution.

Elution: Separation of one material from another by washing or by dissolving one in a solvent in which the other is not soluble.

Elutriate: To separate a coarse, insoluble powder from a finer one by suspending them in water and pouring off the finer powder from the upper part of the fluid.

Emission spectrometry: A rapid analytical technique based on measurement of the characteristic radiation emitted by thermally or electrically excited atoms or ions.

Emphysema: An anatomic alteration of the lung, characterized by abnormal enlargement of air spaces distal to the terminal bronchioles, due to dilation or destructive changes in the alveolar walls.

Emphysematous lesions: A wound or injury to the lung as a result of emphysema.

Empirical modeling: Characterization and description of a phenomena based on experience or observation.

Encephalitis: Inflammation of the brain.

Endoplasmic reticulum: An elaborate membrane structure extending from the nuclear membrane or eucaryotic cells to the cytoplasmic membrane.

Endothelium: A layer of flat cells lining especially blood and lymphatic vessels.

Entropy: A measure of disorder or randomness in a system. Low entropy is associated with highly ordered systems.

Enzyme: Any of numerous proteins produced by living cells which catalyze biological reactions.

Enzyme Commission (EC): The International Commission on Enzymes, established in 1956, developed a scheme of classification and nomenclature under which each enzyme is assigned an EC number which identifies it by function.

Eosinophils: Leukocytes (white blood cells) which stain readily with the dye, eosin.

Epidemiology: A study of the distribution and determinants of disease in human population groups.

Epidermis: The outermost living layer of cells of any organism.

Epididymal fat pads: The fatty tissue located near the epididymis. The epididymis is the first convoluted portion of the excretory duct of the testis.

Epiphyte: A plant growing on another plant but obtaining food from the atmosphere.

Epithelial: Relating to epithelium, the membranous cellular layer which covers free surfaces or lines tubes or cavities of an animal body, which encloses, protects, secretes, excretes and/or assimilates.

Erosion corrosion: Acceleration or increase in rate of deterioration or attack on a metal because of relative movement between a corrosive fluid and the metal surface. Characterized by grooves, gullies, or waves in the metal surface.

Erythrocyte: A mature red blood cell.

Escherichia coli: A short, gram-negative, rod-shaped bacteria common to the human intestinal tract. A frequent cause of infections in the urogenital tract.

Esophageal: Relating to the portion of the digestive tract between the pharynx and the stomach.

Estrus: That portion or phase of the sexual cycle of female animals characterized by willingness to permit coitus.

Estrus cycle: The series of physiologic uterine, ovarian and other changes that occur in higher animals.

Etiolation: Paleness and/or altered development resulting from the absence of light.

Etiology: The causes of a disease or condition; also, the study of causes.

Eucaryotic: Pertaining to those cells having a well-defined nucleus surrounded by a double-layered membrane.

Euthrophication: Elevation of the level of nutrients in a body of water, which can contribute to accelerated plant growth and filling.

Excited state: A state of higher electronic energy than the ground state, usually a less stable one.

Expiratory (maximum) flow rate: The maximum rate at which air can be expelled from the lungs.

Exposure level: Concentration of a contaminant to which an individual or a population is exposed.

Extinction coefficient: A measure of the space rate of diminution, or extinction, of any transmitted light, thus, it is the attenuation coefficient applied to visible radiation.

Extramedullary hematopoiesis: The process of formation and development of the various types of blood cells and other formed elements not including that occurring in bone marrow.

Extravasate: To exclude from or pass out of a vessel into the tissues; applies to urine, lymph, blood and similar fluids.

Far ultraviolet: Radiation in the range of wavelengths from 100 to 190 nanometers.

Federal Reference Method (FRM): For NO_2, the EPA-approved analyzers based on the gas-phase chemiluminescent measurement principle and associated calibration procedures; regulatory specifications prescribed in Title 40, Code of Federal Regulations, Part 50, Appendix F.

Fenestrae: Anatomical aperatures often closed by a membrane.

Fiber: A fine, threadlike piece, as of cotton, jute, or asbestos.

Fiber-reactive dye: A water-soluble dyestuff which reacts chemically with the cellulose in fibers under alkaline conditions; the dye contains two chlorine atoms which combine with the hydroxyl groups of the cellulose.

Fibrin: A white insoluble elastic filamentous protein derived from fibrinogen by the action of thrombin, especially in the clotting of blood.

Fibroadenoma: A benign neoplasm derived from glandular epithelium, involving proliferating fibroblasts, cells found in connective tissue.

Fibroblast: An elongated cell with cytoplasmic processes present in connective tissue, capable of forming collagen fibers.

Fibrosis: The formation of fibrous tissue, usually as a reparative or reactive process and not as a normal constituent of an organ or tissue.

Flocculation: Separation of material from a solution or suspension by reaction with a flocculant to create fluffy masses containing the material to be removed.

Fly ash: Fine, solid particles of noncombustible ash carried out of a bed of solid fuel by a draft.

Folded-path optical system: A long (e.g., 8-22 m) chamber with multiple mirrors at the ends which can be used to reflect an infrared beam through an ambient air sample many times; a spectrometer can be used with such a system to detect trace pollutants at very low levels.

Forced expiratory flow (FEF): The rate at which air can be expelled from the lungs; see expiratory flow rate.

Forced expiratory volume (FEV): The maximum volume of air that can be expired in a specific time interval when starting from maximal inspiration.

Forced vital capacity (FVC): The greatest volume of air that can be exhaled from the lungs under forced conditions after a maximum inspiration.

Fractional threshold concentration: The portion of the concentration at which an event or a response begins to occur, expressed as a fraction.

Free radical: Any of a variety of highly-reactive atoms or molecules characterized by having an unpaired electron.

Fritted bubbler: A porous glass device used in air pollutant sampling systems to introduce small bubbles into solution.

Functional residual capacity: The volume of gas remaining in the lungs at the end of a normal expiration. It is the sum of expiratory reserve volume and residual volume.

Gas exchange: Movement of oxygen from the alveoli into the pulmonary capillary blood as carbon dioxide enters the alveoli from the blood.

Gas chromatography (GC): A method of separating and analyzing mixtures of chemical substances. A flow of gas causes the components of a mixture to migrate differentially from a narrow starting zone in a special porous, insoluble sorptive medium. The pattern formed by zones of separated pigments and of colorless substances in this process is called a chromatogram, and can be analyzed to obtain the concentration of identified pollutants.

Gas-liquid chromatography: A method of separating and analyzing volatile organic compounds, in which a sample is vaporized and swept through a column filled with solid support material covered with a nonvolatile liquid. Components of the sample can be identified and their concentrations determined by analysis of the characteristics of their retention in the column, since compounds have varying degrees of solubility in the liquid medium.

Gastric juice: A thin watery digestive fluid secreted by glands in the mucous membrane of the stomach.

Gastroenteritis: Inflammation of the mucous membrane of stomach and intestine.

Genotype: The type of genes possessed by an organism.

Geometric mean: An estimate of the average of a distribution. Specifically, the nth root of the product of n observations.

Geometric standard deviation: A measure of variability of a distribution. It is the antilogarithm of the standard deviation of the logarithms of the observations.

Globulins (a, b, q): A family of proteins precipitated from plasma (or serum) by half-saturation with ammonium sulfate, or separable by electrophoresis. The main groups are the a, b, q fractions, differing with respect to associated lipids and carbohydrates and in their content of antibodies (immunoglobulins).

Glomular nephrotic syndrome: Dysfunction of the kidneys characterized by excessive protein loss in the urine, accumulation of body fluids and alteration in albumin/globulin ratio.

Glucose: A sugar which is a principal source of energy for man and other organisms.

Glucose-6-phosphate dehydrogenase: An enzyme (EC 1.1.1.49) catalyzing the dehydrogenation of glucose-6-phosphate to 6-phosphogluconolactone.

Glutamic-oxaloacetic transaminase (SGOT): An enzyme (EC 2.6.1.1) whose serum level increases in myocardial infarction and in diseases involving destruction of liver cells. Also known as aspartate aminotransferase.

Glutamic-pyruvic transaminase (SGPT): Now known as alanine aminotransferase (EC 2.6.1.2), the serum levels of this enzyme are used in liver function tests.

Glutathione (GSH): A tripeptide composed of glycine, cystine, and glutamic acid.

Glutathione peroxidase: An enzyme (EC 1.11.1) which catalyzes the destruction of hydroperoxides formed from fatty acids and other substances. Protects tissues from oxidative damage. It is a selenium-containing protein.

Glutathione reductase: The enzyme (EC 1.6.4.2) which reduces the oxidized form of glutathione.

Glycolytic pathway: The biochemical pathway by which glucose is converted to lactic acid in various tissues, yielding energy as a result.

Glycoside: A type of chemical compound formed from the condensation of a sugar with another chemical radical via a hemiacetal linkage.

Goblet cells: Epithelial cells that have been distended with mucin and when this is discharged as mucus, a goblet-shaped shell remains.

Golgi apparatus: A membrane system involved with secretory functions and transport in a cell. Also known as a dictyosome.

Grana: The lamellar stacks of chlorophyll-containing material in plant chloroplasts.

Griege carpet: A carpet in its unfinished state, i.e. before it has been scoured and dyed. The term also is used for woven fabrics in the unbleached and unfinished state.

Ground state: The state of minimum electronic energy of a molecule or atom.

Guanylate cyclase (GC): An enzyme (EC 4.6.2.1) catalyzing the transformation of guanosine triphosphate to guanosine 3':5'-cyclic phosphate.

H-Thymidine: Thymine deoxyribonucleoside: One of the four major nucleosides in DNA. ^{3}H-thymidine has been uniformly labeled with tritium, a radioactive form of hydrogen.

Haze: Fine dust, smoke or fine vapor reducing transparency of air.

Hemagglutination: The agglutination of red blood cells. Can be used as as a measurement of antibody concentration.

Hematocrit: The percentage of the volume of a blood sample occupied by cells.

Hematology: The medical specialty that pertains to the blood and blood-forming tissues.

Hemochromatosis: A disease characterized by pigmentation of the skin possibly due to inherited excessive absorption of iron.

Hemoglobin (Hb): The red, respiratory protein of the red blood cells, hemoglobin transports oxygen from the lungs to the tissues as oxy-hemoglobin (HbO_2) and returns carbon dioxide to the lungs as hemoglobin carbamate, completing the respiratory cycle.

Hemolysis: Alteration or destruction of red blood cells, causing hemoglobin to be released into the medium in which the cells are suspended.

Hepatectomy: Complete removal of the liver in an experimental animal.

Hepatic: Relating to the liver.

Hepatocyte: A liver cell.

Heterogeneous process: A chemical reaction involving reactants of more than one phase or state, such as one in which gases are absorbed into aerosol droplets, where the reaction takes place.

Heterologous: A term referring to donor and recipient cellular elements from different organisms, such as red blood cells from sheep and alveolar macrophage from rabbits.

Hexose monophosphate shunt: Also called the phosphogluconate oxidative pathway of glucose metabolism which affords a total combustion of glucose independent of the citric acid cycle. It is the important generator of NADPH necessary for synthesis of fatty acids and the operation of various enzymes. It serves as a source of ribose and 4- and 7-carbon sugars.

High-volume sampler (Hi-vol): Device for taking a sample of the particulate content of a large amount of air, by drawing air through a fiber filter at a typical rate of 2,000 m^3/24 hr (1.38 m^3/min), or as high as 2,880 m^3/24 hr (2 m^3/min).

Histamine: An amine derived from the amino acid, histidine. It is a powerful stimulant of gastric secretion and a constrictor of bronchial smooth muscle. It is a vasodilator and causes a fall in blood pressure.

Homogenate: Commonly refers to tissue ground into a creamy consistency in which the cell structure is disintegrated.

Host defense mechanism: Inherent means by which a biologic organism protects itself against infection, such as antibody formation, macrophage action, ciliary action, etc.

Host resistance: The resistance exhibited by an organism, such as man, to an infecting agent, such as a virus or bacteria.

Humoral: Relating to the extracellular fluids of the body, blood and lymph.

Hybrid: An organism descended from parents belonging to different varieties or species.

Hydrocarbons: A vast family of compounds containing carbon and hydrogen in various combinations; found especially in fossil fuels. Some contribute to photochemical smog.

Hydrolysis: Decomposition involving splitting of a bond and addition of the H and OH parts of water to the two sides of the split bond.

Hydrometeor: A product of the condensation of atmospheric water vapor (e.g. fog, rain, hail, snow).

Hydroxyproline: An amino acid found among the hydrolysis products of collagen.

Hygroscopic: Pertaining to a marked ability to accelerate the condensation of water vapor.

Hyperplasia: Increase in the number of cells in a tissue or organ excluding tumor formation.

Hyperplastic: Relating to hyperplasia; an increase in the number of cells.

Hypertrophy: Increase in the size of a tissue element, excluding tumor formation.

Hypertension: Abnormally elevated blood pressure.

Hypolimnia: Portions of a lake below the thermocline, in which water is stagnant and uniform in temperature.

Hypoxia: A lower than normal amount of oxygen in the air, blood or tissues.

Immunoglobulin (Ig): A class of structurally related proteins consisting of two pairs of polypeptide chains. Antibodies are Ig's and all Ig's probably function as antibodies.

Immunoglobulin A (IgA): A type of antibody which comprises approximately 10 to 15 percent of the total amount of antibodies present in normal serum.

Immunoglobulin G (IgG): A type of antibody which comprises approximately 80 percent of the total amount of antibodies present in normal serum. Subfractions of IgG are fractions G_1, and G_2.

Immunoglobulin M (IgM): A type of antibody which comprises approximately 5 to 10 percent of the total amount of antibodies present in normal serum.

Impaction: An impinging or striking of one object against another; also, the force transmitted by this act.

Impactor: An instrument which collects samples of suspended particulates by directing a stream of the suspension against a surface, or into a liquid or a void.

Index of proliferation: Ratio of promonocytes to polymorphic monocytes in the blood.

Infarction: Sudden insufficiency of arterial or venous blood supply due to emboli, thrombi, or pressure.

Infectivity model: A testing system in which the susceptibility of animals to airborne infectious agents with and without exposure to air pollutants is investigated to produce information related to the possible effects of the pollutant on man.

Inflorescence: The arrangement and development of flowers on an axis; also, a flower cluster or a single flower.

Influenza A_2/Taiwan Virus: An infectious viral disease, believed to have originated in Taiwan, characterized by sudden onset, chills, fevers, headache, and cough.

Infrared: Light invisible to the human eye, between the wavelengths of 7×10^{-7} and 10^{-3} m (7000 and 10,000,000 A).

Infrared laser: A device that utilizes the natural oscillations of atoms or molecules to generate coherent electromagnetic radiation in the infrared region of the spectrum.

Infrared spectrometer: An instrument for measuring the relative amounts of radiant energy in the infrared region of the spectrum as a function of wavelength.

Ingestion: To take in for digestion.

In situ: In the natural or original position.

Instrumental averaging time: The time over which a single sample or measurement is taken, resulting in a measurement which is an average of the actual concentrations over that period.

Insult: An injury or trauma.

Intercostal: Between the ribs, especially of a leaf.

Interferant: A substance which a measurement method cannot distinguish completely from the one being measured, which therefore can cause some degree of false response or error.

Interferon: A macromolecular substance produced in response to infection with active or inactivated virus, capable of inducing a state of resistance.

Intergranular corrosion: A type of corrosion which takes place at and adjacent to grain boundaries, with relatively little corrosion of the grains.

Interstitial edema: An accumulation of an excessive amount of fluids in a space within tissues.

Interstitial pneumonia: A chronic inflammation of the interstitial tissue of the lung, resulting in compression of air cells.

Intraluminal mucus: Mucus that collects within any tubule.

Intraperitoneal injection: An injection of material into the serous sac that lines the abdominal cavity.

In utero: Within the womb; not yet born.

In vitro: Refers to experiments conducted outside the living organism.

In vivo: Refers to experiments conducted within the living organism.

Irradiation: Exposure to any form of radiation.

Ischemia: Local anemia due to mechanical obstruction (mainly arterial narrowing) of the blood supply.

Isoenzymes: Also called isozymes. One of a group of enzymes that are very similar in catalytic properties, but may be differentiated by variations in physical properties, such as isoelectric point or electrophoretic mobility. Lactic acid dehydrogenase is an example of an enzyme having many isomeric forms.

Isopleth: A line on a map or chart connecting points of equal value.

Jacobs-Hochheiser method: The original Federal Reference Method for NO_2, currently unacceptable for air pollution work.

Klebsiella pneumoniae: A species of rod-shaped bacteria found in soil, water, and in the intestinal tract of man and other animals. Certain types may be causative agents in pneumonia.

Kyphosis: An abnormal curvature of the spine, with convexity backward.

Lactate: A salt or ester of lactic acid.

Lactic acid (lactate) dehydrogenase (LDH): An enzyme (EC 1.1.1.27) with many isomeric forms which catalyzes the oxidation of lactate to pyruvate via transfer of H to NAD. Isomeric forms of LDH in the blood are indicators of heart damage.

Lamellar bodies: Arranged in plates or scales. One of the characteristics of Type II alveolar cells.

Lavage fluid: Any fluid used to wash out hollow organs, such as the lung.

Lecithin: Any of several waxy hygroscopic phosphatides that are widely distributed in animals and plants; they form colloidal solutions in water and have emulsifying, wetting and hygroscopic properties.

Legume: A plant with root nodules containing nitrogen fixing bacteria.

Lesion: A wound, injury or other more or less circumscribed pathologic change in the tissues.

Leukocyte: Any of the white blood cells.

Lewis base: A base, defined in the Lewis acid-base concept, is a substance that can donate an electron pair.

Lichens: Perennial plants which are a combination of two plants, an alga and a fungus, growing together in an association so intimate that they appear as one.

Ligand: Those molecules or anions attached to the central atom in a complex.

Light-fastness: The ability of a dye to maintain its original color under natural or indoor light.

Linolenic acid: An unsaturated fatty acid essential in nutrition.

Lipase: An enzyme that accelerates the hydrolysis or synthesis of fats or the breakdown of lipoproteins.

Lipids: A heterogeneous group of substances which occur widely in biological materials. They are characterized as a group by their extractability in nonpolar organic solvents.

Lipofuscin: Brown pigment granules representing lipid-containing residues of lysosomal digestion. Proposed to be an end product of lipid oxidation which accumulates in tissue.

Lipoprotein: Complex or protein containing lipid and protein.

Loading rate: The amount of a nutrient available to a unit area of body of water over a given period of time.

Locomotor activity. Movement of an organism from one place to another of its own volition.

Long-pathlength infrared absorption: A measurement technique in which a system of mirrors in a chamber is used to direct an infrared beam through a sample of air for a long distance (up to 2 km); the amount of infrared absorbed is measured to obtain the concentrations of pollutants present.

Lung compliance (C_L): The volume change produced by an increase in a unit change in pressure across the lung, i.e., between the pleural surface and the mouth.

Lycra: A spandex textile fiber created by E. I. du Pont de Nemours & Co., Inc., with excellent tensile strength, a long flex life and high resistance to abrasion and heat degradation. Used in brassieres, foundation garments, surgical hosiery, swim suits and military and industrial uses.

Lymphocytes: White blood cells formed in lymphoid tissue throughout the body, they comprise about 22 to 28 percent of the total number of leukocytes in the circulating blood and function in immunity.

Lymphocytogram: The ratio, in the blood, of lymphocyte with narrow cytoplasm to those with broad cytoplasm.

Lysosomes: Organelles found in cells of higher organisms that contain high concentrations of degradative enzymes and are known to destroy foreign substances that cells engulf by pinocytosis and phyocytosis. Believed to be a major site where proteins are broken down.

Lysozymes: Lytic enzymes destructive to cell walls of certain bacteria. Present in some body fluids, including tears and serum.

Macaca speciosa: A species of monkeys used in research.

Macrophage: Any large, ameboid, phagocytic cell having a nucleus without many lobes, regardless of origin.

Malaise: A feeling of general discomfort or uneasiness, often the first indication of an infection or disease.

Malate dehydrogenase: An enzyme (EC 1.1.1.37) with at least six isomeric forms that catalyze the dehydrogenation of malate to oxaloacetate or its decarboxylation (removal of a CO_2 group) to pyruvate. Malate, oxaloacetate, and pyruvate are intermediate components of biochemical pathways.

Mannitol: An alcohol derived from reduction of the sugar, fructose. Used in renal function testing to measure glomerular (capillary) filtration.

Manometer: An instrument for the measurement of pressure of gases or vapors.

Mass median diameter (MMD): Geometric median size of a distribution of particles based on weight.

Mass spectrometry (MS): A procedure for identifying the various kinds of particles present in a given substance, by ionizing the particles and subjecting a beam of the ionized particles to an electric or magnetic field such that the field deflects the particles in angles directly proportional to the masses of the particles.

Maximum flow (V_{max}): Maximum rate or expiration, usually expressed at 50 or 25 percent of vital capacity.

Maximum mid-expiratory flow rate (MMFR): The mean rate of expiratory gas flow between 25 and 75 percent of the forced expiratory vital capacity.

Mean (arithmetic): The sum of observations divided by sample size.

Median: A value in a collection of data values which is exceeded in magnitude by one-half the entries in the collection.

Mesoscale: Of or relating to meteorological phenomena from 1 to 100 kilometers in horizontal extent.

Messenger RNA: A type of RNA which conveys genetic information encoded in the DNA to direct protein synthesis.

Metaplasia: The abnormal transformation of an adult, fully differentiated tissue of one kind into a differentiated tissue of another kind.

Metaproterenol: A bronchodilator used for the treatment of bronchial asthma.

Metastases: The shifting of a disease from one part of the body to another; the appearance of neoplasms in parts of the body remote from the seat of the primary tumor.

Meteorology: The science that deals with the atmosphere and its phenomena.

Methemoglobin: A form of hemoglobin in which the normal reduced state of iron (Fe^{2+}) has been oxidized to Fe^{3+}. It contains oxygen in firm union with ferric (Fe^{3+}) iron and is not capable of exchanging oxygen in normal respiratory processes.

Methimazole: An anti-thyroid drug similar in action to propylthiouracil.

Methyltransferase: Any enzyme transferring methyl groups from one compound to another.

Microcoulometric: Capable of measuring millionths of coulombs used in electrolysis of a substance, to determine the amount of a substance in a sample.

Microflora: A small or strictly localized plant.

Micron: One-millionth of a meter.

Microphage: A small phagocyte; a polymorphonuclear leukocyte that is phagocytic.

Millimolar: One-thousandth of a molar solution. A solution of one-thousandth of a mole (in grams) per liter.

Minute volume: The minute volume of breathing; a product of tidal volume times the respiratory frequency in one minute.

Mitochondria: Organelles of the cell cytoplasm which contain enzymes active in the conservation of energy obtained in the aerobic part of the breakdown of carbohydrates and fats, in a process called respiration.

Mobile sources: Automobiles, trucks and other pollution sources which are not fixed in one location.

Modacrylic fiber: A manufactured fiber in which the fiber-forming substance is any long chain synthetic polymer composed of less than 85 percent but at least 35 percent by weight of acrylonitrite units.

Moeity: One of two or more parts into which something is divided.

Mole: The mass, in grams, numerically equal to the molecular weight of a substance.

Molecular correlation spectrometry: A spectrophotometric technique which is used to identify unknown absorbing materials and measure their concentrations by using preset wavelengths.

Molecular weight: The weight of one molecule of a substance obtained by adding the gram-atomic weights of each of the individual atoms in the substance.

Monocyte: A relatively large mononuclear leukocyte, normally constituting 3 to 7 percent of the leukocytes of the circulating blood.

Mordant: A substance which acts to bind dyes to a textile fiber of fabric.

Morphological: Relating to the form and structure of an organism or any of its parts.

Moving average: A procedure involving taking averages over a specific period prior to and including a year in question, so that successive averaging periods overlap; e.g. a three-year moving average would include data from 1967 through 1969 for the 1969 average and from 1968 through 1970 for 1970.

Mucociliary clearance: Removal of materials from the upper respiratory tract via ciliary action.

Mucociliary transport: The process by which mucus is transported, by ciliary action, from the lungs.

Mucosa: The mucous membrane; it consists of epithelium, lamina propria and, in the digestive tract, a layer of smooth muscle.

Mucous membrane: A membrane secreting mucus which lines passages and cavities communicating with the exterior of the body.

Murine: Relating to mice.

Mutagen: A substance capable of causing, within an organism, biological changes that affect potential offspring through genetic mutation.

Mutagenic: Having the power to cause mutations. A mutation is a change in the character of a gene (a sequence of base pairs in DNA) that is perpetuated in subsequent divisions of the cell in which it occurs.

Myocardial infarction: Infarction of any area of the heart muscle usually as a result of occlusion of a coronary artery.

Nares: The nostrils.

Nasopharyngeal: Relating to the nasal cavity and the pharynx (throat).

National Air Surveillance Network (NASN): Network of monitoring stations for sampling air to determine extent of air pollution; established jointly by federal and state governments.

Near ultraviolet: Radiation of the wavelengths 2000-4000 Angstroms.

Necrosis: Death of cells that can discolor areas of a plant or kill the entire plant.

Necrotic: Pertaining to the pathologic death of one or more cells, or of a portion of tissue or organ, resulting from irreversible damage.

Neonate: A newborn.

Neoplasm: An abnormal tissue that grows more rapidly than normal; synonymous with tumor.

Neoplasia: The pathologic process that results in the formation and growth of a tumor.

Neutrophil: A mature white blood cell formed in bone marrow and released into the circulating blood, where it normally accounts for 54 to 65 percent of the total number of leukocytes.

Ninhydrin: An organic reagent used to identify amino acids.

Nitramine: A compound consisting of a nitrogen attached to the nitrogen of amine.

Nitrate: A salt or ester of nitric acid (NO_3^-).

Nitrification: The principal natural source of nitrate in which ammonium (NH_4+) ions are oxidized to nitrites by specialized microorganisms. Other organisms oxidize nitrites to nitrates.

Nitrite: A salt or ester of nitrous acid ($NO_2^=$).

Nitrocellulose: Any of several esters of nitric acid formed by its action on cellulose, used in explosives, plastics, varnishes and rayon; also called cellulose nitrate.

Nitrogen cycle: Refers to the complex pathways by which nitrogen-containing compounds are moved from the atmosphere into organic life, into the soil, and back to the atmosphere.

Nitrogen fixation: The metabolic assimilation of atmospheric nitrogen by soil microorganisms, which becomes available for plant use when the microorganisms die; also, industrial conversion of free nitrogen into combined forms used in production of fertilizers and other products.

Nitrogen oxide: A compound composed of only nitrogen and oxygen. Components of photochemical smog.

Nitrosamine: A compound consisting of a nitrosyl group connected to the nitrogen of an amine.

Nitrosation: Addition of a nitrosyl group.

N-Nitroso compounds: Compounds carrying the functional nitrosyl group.

Nitrosyl: A group composed of one oxygen and one nitrogen atom (-N=O).

Nitrosylhemoglobin (NOHb): The red, respiratory protein of erythrocytes to which a nitrosyl group is attached.

N/P Ratio: Ratio of nitrogen to phosphorous dissolved in lake water, important due to its effect on plant growth.

Nucleolus: A small spherical mass of material within the substance of the nucleus of a cell.

Nucleophilic: Having an affinity for atomic nuclei; electron-donating.

Nucleoside: A compound that consists of a purine or pyrimidine base combined with deoxyribose or ribose and found in RNA and DNA.

5'-Nucleotidase: An enzyme (EC 3.1.3.5) which hydrolyzes nucleoside 5'-phosphates into phosphoric acid (H_3PO_4) and nucleosides.

Nucleotide: A compound consisting of a sugar (ribose or deoxyribose), a base (a purine or a pyrimidine), and a phosphate; a basic structural unit of RNA and DNA.

Nylon: A generic name chosen by E. I. du Pont de Nemours & Co., Inc. for a group of protein-like chemical products classed as synthetic linear polymers; two main types are Nylon 6 and Nylon 66.

Occlusion: A point which an opening is closed or obstructed.

Olefin: An open-chain hydrocarbon having at least one double bond.

Olfactory: Relating to the sense of smell.

Olfactory epithelium: The inner lining of the nose and mouth which contains neural tissue sensitive to smell.

Oligotrophic: A body of water deficient in plant nutrients; also generally having abundant dissolved oxygen and no marked stratification.

Oribitals: Areas of high electron density in an atom or molecule.

Orlon: An acrylic fiber produced by E. I. du Pont de Nemours and Co., Inc., based on a polymer of acrylonitrite; used extensively for outdoor uses, it is resistant to chemicals and withstands high temperatures.

Osteogenic osteosarcoma: The most common and malignant of bone sarcomas (tumors). It arises from bone-forming cells and affects chiefly the ends of long bones.

Ovarian primordial follicle: A spheroidal cell aggregation in the ovary in which the primordial oocyte (immature female sex cell) is surrounded by a single layer of flattened follicular cells.

Oxidant: A chemical compound which has the ability to remove electrons from another chemical species, thereby oxidizing it; also, a substance containing oxygen which reacts in air to produce a new substance, or one formed by the action of sunlight on oxides of nitrogen and hydrocarbons.

Oxidation: An ion or molecule undergoes oxidation by donating electrons.

Oxidative deamination: Removal of the NH_2 group from an amino compound by reaction with oxygen.

Oxidative phosphorylation: The mitochondrial process by which "high-energy" phosphate bonds form from the energy released as a result of the oxidation of various substrates. Principally occurs in the tricarboxylic acid pathway.

Oxyhemoglobin: Hemoglobin in combination with oxygen. It is the form of hemoglobin present in arterial blood.

Ozone layer: A layer of the stratosphere from 20 to 50 km above the earth's surface characterized by high ozone content produced by ultraviolet radiation.

Ozone scavenging: Removal of O_3 from ambient air or plumes by reaction with NO, producing NO_2 and O_2.

Paired electrons: Electrons having opposite intrinsic spins about their own axes.

Parenchyma: The essential and distinctive tissue of an organ or an abnormal growth, as distinguished from its supportive framework.

Parenchymal: Referring to the distinguishing or specific cells of a gland or organ.

Partial pressure: The pressure exerted by a single component in a mixture of gases.

Particulates: Fine liquid or solid particles such as dust, smoke, mist, fumes or smog, found in the air or in emissions.

Pascal: A unit of pressure in the International System of Units. One pascal is equal to 7.4×10^{-3} torr. The pascal is equivalent to one newton per square meter.

Pathogen: Any virus, microorganism, or other substance causing disease.

Pathophysiological: Derangement of function seen in disease; alteration in function as distinguished from structural defects.

Peptide bond: The bond formed when two amino acids react with each other.

Percentiles: The percentage of all observations exceeding or preceding some point; thus, 90th percentile is a level below which will fall 90 percent of the observations.

Perfusate: A liquid, solution or colloidal suspension that has been passed over a special surface or through an appropriate structure.

Perfusion: Artificial passage of fluid through blood vessels.

Permanent-press fabrics: Fabrics in which applied resins contribute to the easy care and appearance of the fabric and to the crease and seam flatness by reacting with the cellulose on pressing after garment manufacture.

Permeation tube: A tube which is selectively porous to specific gases.

Peroxidation: Refers to the process by which certain organic compounds are converted to peroxides.

Peroxyacetyl nitrate (PAN): Pollutant created by action of sunlight on hydrocarbons and NO_x in the air; an ingredient of photochemical smog.

pH: A measure of the acidity or alkalinity of a material, liquid, or solid. pH is represented on a scale of 0 to 14 with 7 being a neutral state, 0 most acid, and 14 most alkaline.

Phagocytosis: Ingestion, by cells such as macrophages, of other cells, bacteria, foreign particles, etc.; the cell membrane engulfs solid or liquid particles which are drawn into the cytoplasm and digested.

Phenotype: The observable characteristics of an organism, resulting from the interaction between an individual genetic structure and the environment in which development takes place.

Phenylthiourea: A crystalline compound, $C_7H_8N_2S$, that is bitter or tasteless depending on a single dominant gene in the tester.

Phlegm: Viscid mucus secreted in abnormal quantity in the respiratory passages.

Phosphatase: Any of a group of enzymes that liberate inorganic phosphate from phosphoric esters (E.C. sub-subclass 3.1.3).

Phosphocreatine kinase: An enzyme (EC 2.7.3.2) catalyzing the formation of creatine and ATP, its breakdown is a source of energy in the contraction of muscle; also called creatine phosphate.

Phospholipid: A molecule consisting of lipid and phosphoric acid group(s). An example is lecithin. Serves as an important structural factor in biological membranes.

Photochemical oxidants: Primary ozone, NO_2, PAN with lesser amounts of other compounds formed as products of atmospheric reactions involving organic pollutants, nitrogen oxides, oxygen, and sunlight.

Photochemical smog: Air pollution caused by chemical reaction of various airborne chemicals in sunlight.

Photodissociation: The process by which a chemical compound breaks down into simpler components under the influence of sunlight or other radiant energy.

Photolysis: Decomposition upon irradiation by sunlight.

Photomultiplier tube: An electron multiplier in which electrons released by photoelectric emission are multiplied in successive stages by dynodes that produce secondary emissions.

Photon: A quantum of electromagnetic energy.

Photostationary: A substance or reaction which reaches and maintains a steady state in the presence of light.

Photosynthesis: The process in which green parts of plants, when exposed to light under suitable conditions of temperature and water supply, produce carbohydrates using atmospheric carbon dioxide and releasing oxygen.

Phytotoxic: Poisonous to plants.

Phytoplankton: Minute aquatic plant life.

Pi (Π) bonds: Bonds in which electron density is not symmetrical about a line joining the bonded atoms.

Pinocytotic: Refers to the cellular process (pinocytosis) in which the cytoplasmic membrane forms invaginations in the form of narrow channels leading into the cell. Liquids can flow into these channels and the membrane pinches off pockets that are incorporated into the cytoplasm and digested.

Pitting: A form of extremely localized corrosion that results in holes in the metal. One of the most destructive forms of corrosion.

Pituary: A stalk-like gland near the base of the brain which is attached to the hypothalmus. The anterior portion is a major repository for for hormones that control growth, stimulate other glands, and regulate the reproductive cycle.

Placenta: The organ in the uterus that provides metabolic interchange between the fetus and mother.

Plasmid: Replicating unit, other than a nucleus gene, that contains nucleoprotein and is involved in various aspects of metabolism in organisms; also called paragenes.

Plasmolysis: The dissolution of cellular components, or the shrinking of plant cells by osmotic loss of cytoplasmic water.

Plastic: A plastic is one of a large group of organic compounds synthesized from cellulose, hydrocarbons, proteins or resins and capable of being cast, extruded, or molded into various shapes.

Plasticizer: A chemical added to plastics to soften, increase malleability or to make more readily deformable.

Platelet (blood): An irregularly-shaped disk with no definite nucleus; about one-third to one-half the size of an erythrocyte and containing no hemoglobin. Platelets are more numerous than leukocytes, numbering from 200,000 to 300,000 per cu. mm. of blood.

Plethysmograph: A device for measuring and recording changes in volume of a part, organ or the whole body; a body plethysmograph is a chamber apparatus surrounding the entire body.

Pleura: The serous membrane enveloping the lungs and lining the walls of the chest cavity.

Plume: Emission from a flue or chimney, usually distributed stream-like downwind of the source, which can be distinguished from the surrounding air by appearance or chemical characteristics.

Pneumonia (interstitial): A chronic inflammation of the interstitial tissue of the lung, resulting in compression of the air cells. An acute, infectious disease.

Pneumonocytes: A nonspecific term sometimes used in referring to types of cells characteristic of the respiratory part of the lung.

Podzol: Any of a group of zonal soils that develop in a moist climate, especially under coniferous or mixed forest.

Point source: A single stationary location of pollutant discharge.

Polarography: A method of quantitative or qualitative analysis based on current-voltage curves obtained by electrolysis of a solution with steadily increasing voltage.

Pollution gradient: A series of exposure situations in which pollutant concentrations range from high to low.

Polyacrylonitrile: A polymer made by reacting ethylene oxide and hydrocyanic acid. Dynel and Orlon are examples.

Polyamides: Polymerization products of chemical compounds which contain amino ($-NH_2$) and carboxyl (-COOH) groups. Condensation reactions between the groups form amides ($-CONH_2$). Nylon is an example of a polyamide.

Polycarbonate: Any of various tough transparent thermoplastics characterized by high impact strength and high softening temperature.

Polycythemia: An increase above the normal in the number of red cells in the blood.

Polyester fiber: A man-made or manufactured fiber in which the fiber-forming substance is any long-chain synthetic polymer composed of at least 85 percent by weight of an ester of a dihydric alcohol and terephthalic acid. Dacron is an example.

Polymer: A large molecule produced by linking together many like molecules.

Polymerization: In fiber manufacture, converting a chemical monomer (simple molecule) into a fiber-forming material by joining many like molecules into a stable, long-chain structure.

Polymorphic monocyte: Type of leukocyte with a multi-lobed nucleus.

Polymorphonuclear leukocytes: Cells which represent a secondary non-specific cellular defense mechanism. They are transported to the lungs from the bloodstream when the burden handled by the alveolar macrophages is too large.

Polysaccharides: Polymers made up of sugars. An example is glycogen which consists of repeating units of glucose.

Polystyrene: A thermoplastic plastic which may be transparent, opaque, or translucent. It is light in weight, tasteless and odorless, it also is resistant to ordinary chemicals.

Polyurethane: Any of various polymers that contain NHCOO linkages and are used especially in flexible and rigid foams, elastomers and resins.

Pores of Kohn: Also known as interalveolar pores; pores between air cells. Assumed to be pathways for collateral ventilation.

Precipitation: Any of the various forms of water particles that fall from the atmosphere to the ground, rain, snow, etc.

Precursor: A substance from which another substance is formed; specifically, one of the anthropogenic or natural emissions or atmospheric constituents which reacts under sunlight to form secondary pollutants comprising photochemical smog.

Probe: In air pollution sampling, the tube or other conduit extending into the atmosphere to be sampled, through which the sample passes to treatment, storage and/or analytical equipment.

Proline: An amino acid, $C_5H_9NO_2$, that can be synthesized from glutamate by animals.

Promonocyte: An immature monocyte not normally seen in the circulating blood.

Proteinuria: The presence of more than 0.3 gm of urinary protein in a 24-hour urine collection.

Pulmonary: Relating to the lungs.

Pulmonary edema: An accumulation of excessive amounts of fluid in the lungs.

Pulmonary lumen: The spaces in the interior of the tubular elements of the lung (bronchioles and alveolar ducts).

Pulmonary resistance: Sum of airway resistance and viscous tissue resistance.

Purine bases: Organic bases which are constituents of DNA and RNA, including adenine and guanine.

Purulent: Containing or forming pus.

Pyrimidine bases: Organic bases found in DNA and RNA. Cytosine and thymine occur in DNA and cytosine and uracil are found in RNA.

QRS: Graphical representation on the electrocardiogram of a complex of three distinct waves which represent the beginning of ventricular contraction.

Quasistatic compliance: Time dependent component of elasticity; compliance is the reciprocal of elasticity.

Rainout: Removal of particles and/or gases from the atmosphere by their involvement in cloud formation (particles act as condensation nuclei, gases are absorbed by cloud droplets), with subsequent precipitation.

Rayleigh scattering: Coherent scattering in which the intensity of the light of wavelength λ, scattered in any direction making an angle with the incident direction, is directly proportional to $1 + \cos^2\theta$ and inversely proportional to λ^4.

Reactive dyes: Dyes which react chemically with cellulose in fibers under alkaline conditions. Also called fiber reactive or chemically reactive dyes.

Reduction: Acceptance of electrons by an ion or molecule.

Reference method (RM): For NO_2, an EPA-approved gas-phase chemiluminescent analyzer and associated calibration techniques; regulatory specifications are described in Title 40, Code of Federal Regulations, Part 50, Appendix F. Formerly, Federal Reference Method.

Residual capacity: The volume of air remaining in the lungs after a maximum expiratory effort; same as residual volume.

Residual volume (RV): The volume of air remaining in the lungs after a maximal expiration. RV = TLC - VC

Resin: Any of various solid or semi-solid amorphous natural organic substances, usually derived from plant secretions, which are soluble in organic solvents but not in water; also any of many synthetic substances with similar properties used in finishing fabrics, for permanent press shrinkage control or water repellency.

Ribosomal RNA: The most abundant RNA in a cell and an integral constituent of ribosomes.

Ribosomes: Discrete units of RNA and protein which are instrumental in the synthesis of proteins in a cell. Aggregates are called polysomes.

Runoff: Water from precipitation, irrigation or other sources that flows over the ground surface to streams.

Sclerosis: Pathological hardening of tissue, especially from overgrowth of fibrous tissue or increase in interstitial tissue.

Selective leaching: The removal of one element from a solid alloy by corrosion processes.

Septa: A thin wall dividing two cavities or masses of softer tissue.

Seromucoid: Pertaining to a mixture of watery and mucinous material such as that of certain glands.

Serum antiprotease: A substance, present in serum, that inhibits the activity of proteinases (enzymes which destroy proteins).

Sigma (s) bonds: Bonds in which electron density is symmetrical about a line joining the bonded atoms.

Silo-filler's disease: Pulmonary lesion produced by oxides of nitrogen produced by fresh silage.

Single breath nitrogen elimination rate: Percentage rise in nitrogen fraction per unit of volume expired.

Single breath nitrogen technique: A procedure in which a vital capacity inspiration of 100 percent oxygen is followed by examination of nitrogen in the vital capacity expirate.

Singlet state: The highly-reactive energy state of an atom in which certain electrons have unpaired spins.

Sink: A reactant with or absorber of a substance.

Sodium arsenite: Na_3AsO_3, used with sodium hydroxide in the absorbing solution of a 24-hour integrated manual method for NO_2.

Sodium dithionite: A strong reducing agent (a supplier of electrons).

Sodium metabisulfite: $Na_2S_2O_5$, used in absorbing solutions of NO_2 analysis methods.

Sorb: To take up and hold by absorption or adsorption.

Sorbent: A substance that takes up and holds another by absorption or adsorption.

Sorbitol dehydrogenase: An enzyme that interconverts the sugars, sorbitol and fructose.

Sorption: The process of being sorbed.

Spandex: A manufactured fiber in which the fiber forming substance is a long chain synthetic elastomer composed of at least 85 percent of a segmented polyurethane.

Spectrometer: An instrument used to measure radiation spectra or to determine wavelengths of the various radiations.

Spectrophotometry: A technique in which visible, UV, or infrared radiation is passed through a substance or solution and the intensity of light transmitted at various wavelengths is measured to determine the spectrum of light absorbed.

Spectroscopy: Use of the spectrometer to determine concentrations of an air pollutant.

Spermatocytes: A cell destined to give rise to spermatozoa (sperm).

Sphingomyelins: A group of phospholipids found in brain, spinal cord, kidney and egg yolk.

Sphygomenometer: An apparatus, consisting of a cuff and a pressure gauge, which is used to measure blood pressure.

Spirometry: Also called pneometry. Testing the air capacity of the lungs with a pneometer.

Spleen: A large vascular organ located on the upper left side of the abdominal cavity. It is a blood-forming organ in early life. It is a storage organ for red corpuscles and because of the large number of macrophages, acts as a blood filter.

Sputum: Expectorated matter, especially mucus or mucopurulent matter expectorated in diseases of the air passages.

Squamous: Scale-like, scaly.

Standard deviation: Measure of the dispersion of values about a mean value. It is calculated as the positive square root of the average of the squares of the individual deviations from the mean.

Standard temperature and pressure: $0^{o}C$, 760 mm mercury.

Staphylococcus aureus: A spherically-shaped, infectious species of bacteria found especially on nasal mucous membrane and skin.

Static lung compliance (C_{Lstat}): Measure of lung's elastic recoil (volume change resulting from change in pressure) with no or insignificant airflow.

Steady state exposure: Exposure to air pollutants whose concentration remains constant for a period of time.

Steroids: A large family of chemical substances comprising many hormones and vitamins and having large ring structures.

Stilbene: An aromatic hydrocarbon $C_{14}H_{12}$ used as a phosphor and in making dyes.

Stoichiometric factor: Used to express the conversion efficiency of a non-quantitative reaction, such as the reaction of NO_2 with azo dyes in air monitoring methods.

Stoma: A minute opening or pore (plural is stomata).

Stratosphere: That region of the atmosphere extending from 11 km above the surface of the earth to 50 km. At 50 km above the earth temperature rises to a maximum of $0^{o}C$.

Streptococcus pyogenes: A species of bacteria found in the human mouth, throat and respiratory tract and in inflammatory exudates, blood stream, and lesions in human diseases. It causes formation of pus or even fatal septicemias.

Stress corrosion cracking: Cracking caused by simultaneous presence of tensile stress and a specific corrosive medium. The metal or alloy is virtually unattached over most of its surface, while fine cracks progress through it.

Strong interactions: Forces or bond energies holding molecules together. Thermal energy will not disrupt the formed bonds.

Sublobular hepatic necrosis: The pathologic death of one or more cells, or of a portion of the liver, beneath one or more lobes.

Succession: The progressive natural development of vegetation towards a climax, during which one community is gradually replaced by others.

Succinate: A salt of succinic acid involved in energy production in the citric acid cycle.

Sulfadiazine: One of a group of sulfa drugs. Highly effective against pneumococcal, staphlococcal, and streptococcal infections.

Sulfamethazine: An antibacterial agent of the sulfonamide group, active against homolytic streptococci, staphytococci, pneumococci and meningococci.

Sulfanilimide: A crystalline sulfonamide ($C_6H_8N_2O_2S$), the amide of sulfanilic acid and parent compound of most sulfa drugs.

Sulfhydryl group: A chemical radical consisting of sulfur and hydrogen which confers reducing potential to the chemical compound to which it is attached (-SH).

Sulfur dioxide (SO_2): Colorless gas with pungent odor released primarily from burning of fossil fuels, such as coal, containing sulfur.

Sulfur dyes: Used only on vegetable fibers, such as cottons. They are insoluble in water and must be converted chemically in order to be soluble. They are resistant (fast) to alkalies and washing and fairly fast to sunlight.

Supernatant: The clear or partially clear liquid layer which separates from the homogenate upon centrifugation or standing.

Surfactant: A substance capable of altering the physiochemical nature of surfaces, such as one used to reduce surface tension of a liquid.

Symbiotic: A close association between two organisms of different species in which at least one of the two benefits.

Synergistic: A relationship in which the combined action or effect of two or more components is greater than that of the components acting separately.

Systolic: Relating to the rhythmical contraction of the heart.

Tachypnea: Very rapid breathing.

Terragram (Tg): One million metric tons, 10^{12} grams.

Teratogenesis: The disturbed growth processes resulting in a deformed fetus.

Teratogenic: Causing or relating to abnormal development of the fetus.

Threshold: The level at which a physiological or psychological effect begins to be produced.

Thylakoid: A membranous lamella of protein and lipid in plant chloroplasts where the photochemical reactions of photosynthesis take place.

Thymidine: A nucleoside ($C_{10}H_{14}N_2O_5$) that is composed of thymine and deoxyribose; occurs as a structural part of DNA.

Tidal volume (V_T): The volume of air that is inspired or expired in a single breath during regular breathing.

Titer: The standard of strength of a volumetric test solution. For example, the titration of a volume of antibody-containing serum with another volume containing virus.

Tocopherol: a-d-tocopherol is one form of Vitamin E prepared synthetically. The a form exhibits the most biological activity. It is an antioxidant and retards rancidity of fats.

Torr: A unit of pressure sufficient to support a 1 mm column of mercury; 760 torr = 1 atmosphere.

Total lung capacity (TLC): The sum of all the compartments of the lung, or the volume of air in the lungs at maximum inspiration.

Total suspended particulates (TSP): Solid and liquid particles present in the atmosphere.

Trachea: Commonly known as the windpipe, a cartilaginous air tube extending from the larnyx (voice box) into the thorax (chest) where it divides, serving as the entrance to each of the lungs.

Transaminase: Aminotransferase; an enzyme transferring an amino group from an a-amino acid to the carbonyl carbon atom of an a-keto acid.

Transmissivity (UV): The percent of ultraviolet radiation passing through a medium.

Transmittance: The fraction of the radiant energy entering an absorbing layer which reaches the layer's further boundary.

Transpiration: The process of the loss of water vapor from plants.

Triethanolamine: An amine, $(HOCH_2CH_2)_3N$, used in the absorbing solution of one analytical method for NO_2.

Troposphere: That portion of the atmosphere in which temperature decreases rapidly with altitude, clouds form, and mixing of air masses by convection takes place. Generally extends to about 7 to 10 miles above the earth's surface.

Type 1 epithelial cells: Squamous cells which provide a continuous lining to the alveolar surface.

Type I pneumonocytes: Pulmonary surface epithelial cells.

Type II pneumonocytes: Great alveolar cells.

Ultraviolet: Light invisible to the human eye of wavelengths between $4\text{x}10^{-7}$ and $5\text{x}10^{-9}$ m (4000 to 50Å).

Urea-formaldehyde resin: A compound composed of urea and formaldehyde in an arrangement that conveys thermosetting properties.

Urobilinogen: One of the products of destruction of blood cells; found in the liver, intestines and urine.

Uterus: The womb; the hollow muscular organ in which the impregnated ovum (egg) develops into the fetus.

Vacuole: A minute space in any tissue.

Vagal: Refers to the vagus nerve. This mixed nerve arises near the medulla oblongata and passes down from the cranial cavity to supply the larynx, lungs, heart, esophagus, stomach, and most of the abdominal viscera.

Valence: The number of electrons capable of being bonded or donated by an atom during bonding.

Van Slyke reactions: Reaction of primary amines, including amino acids, with nitrous acid, yielding molecular nitrogen.

Variance: A measure of dispersion or variation of a sample from its expected value; it is usually calculated as the square root a sum of squared deviations about a mean divided by the sample size.

Vat dyes: Dyes which have a high degree of resistance to fading by light, NO_x and washing. Widely used on cotton and viscose rayon. Colors are brilliant and of almost any shade. The name was originally derived from their application in a vat.

Venezuelan equine encephalomyelitis: A form of equine encephalomyelitis found in parts of South America, Panama, Trinidad, and the United States, and caused by a virus. Fever, diarrhea, and depression are common. In man, there is fever and severe headache after an incubation period of 2 to 5 days.

Ventilatory volume (V_E): The volume of gas exchanged between the lungs and the atmosphere that occurs in breathing.

Villus: A projection from the surface, especially of a mucous membrane.

Vinyl chloride: A gaseous chemical suspected of causing at least one type of cancer. It is used primarily in the manufacture of polyvinyl chloride, a plastic.

Viscose rayon: Filaments of regenerated cellulose coagulated from a solution of cellulose xanthate. Raw materials can be cotton linters or chips of spruce, pine, or hemlock.

Visible region: Light between the wavelengths of 4000-8000 Å.

Visual range: The distance at which an object can be distinguished from background.

Vital capacity: The greatest volume of air that can be exhaled from the lungs after a maximum inspiration.

Vitamin E: Any of several fat-soluble vitamins (tocopherols), essential in nutrition of various vertebrates.

Washout: The capture of gases and particles by falling raindrops.

Weak interactions: Forces, electrostatic in nature, which bind atoms and/or molecules to each other. Thermal energy will disrupt the interaction. Also called van der Waal's forces.

Wet deposition: The process by which atmospheric substances are returned to earth in the form of rain or other precipitation.

Wheat germ lipase: An enzyme, obtained from wheat germ, which is capable of cleaving a fatty acid from a neutral fat; a lipolytic enzyme.

X-ray fluorescence spectrometry: A nondestructive technique which utilizes the principle that every element emits characteristic x-ray emissions when excited by high-energy radiation.

Zeolites: Hydrous silicates analogous to feldspars, occurring in lavas and various soils.

Zooplankton: Minute animal life floating or swimming weakly in a body of water.

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